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ENDOMETRIAL CARCINOMA OF THE OVARY, ARISING IN ENDOMETRIAL TISSUE IN THAT ORGAN *

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Many interesting and important problems have presented themselves to clinicians and pathologists, who have appreciated the frequency of ectopic endometrium-like tissue in the pelvis and have had an opportunity to observe the various lesions in the ovaries and other pelvic structures resulting from this tissue. One of the most interesting problems is the source of these implantation-like lesions occurring on the surface of the various pelvic structures. Are they true implantations derived primarily from uterine or tubal epithelium escaping through the tubes into the peritoneal cavity, as the study of many of these lesions in human beings indicates [1] and as Jacobson,[2] in the autotransplantation of endometrial tissue in rabbits, has experimentally proved possible, or are they derived from scattered localized metaplasias of the serosal mesothelium or from developmental inclusions of portions of the Wolffian body or Müllerian duct? This problem has not yet been definitely solved to the satisfaction of all who are interested in the subject. An exhaustive presentation of the serosal origin of these lesions has recently been published by Lauche.[3] Irrespective of their

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1. Sampson, J. A.: Intestinal Adenomas of Endometrial Type, Arch. Surg. **5**:217-280 (Sept.) 1922; The Life History of Ovarian Hematomas (Hemorrhagic Cysts) of Endometrial (Müllerian) Type, Am. J. Obst. & Gynec. **4**:451-512 (Nov.) 1922; Benign and Malignant Endometrial Implants in the Peritoneal Cavity and Their Relation to Certain Ovarian Tumors, Surg., Gynec. & Obst. **38**:287-311 (March) 1924.

2. Jacobson, V. C.: The Autotransplantation of Endometrial Tissue in the Rabbit, Arch. Surg. **5**:281-300 (Sept.) 1922; Further Studies in Autotransplantation of Endometrial Tissue in the Rabbit, Am. J. Obst. & Gynec. **6**:257-262 (Sept.) 1923.

3. Lauche, Arnold: Die Extrogenitalen heterotopen Epithelwucherungen von Bau der Uterusschleimhaut (Fibroadenomatosis Seroepithelialis), Virchow's Archiv. f. Pathologische Anatomie und Physiologie **243**:298-372 (April) 1923.

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origin, another important problem presents itself, and that is the possibility of the development of cancer in this misplaced endometrial tissue wherever the latter may be situated. If it can be demonstrated that cancer may arise in this tissue and in the lesions resulting from it, the result will be a most rational explanation of the histogenesis of some, and possibly even of many, apparently primary ovarian carcinomas as well as of peritoneal carcinosis of obscure origin. Furthermore, if cancer should arise in any misplaced endometrium-like tissue, it would suggest a true epithelial origin for that tissue.

It is generally accepted that the majority of the benign and malignant epithelial tumors of the ovary arise from the germinal epithelium of that organ, and also that this epithelium as well as that lining the tubes and uterus have a common embryonic origin. An apparent confirmation of the foregoing is the fact that the epithelial lining of many ovarian cysts resembles that of the tubes and uterus (including the cervix), and adenocarcinoma of the ovary often has the same histologic structure as that of the tubes and uterus. If this hypothesis as to the origin of ovarian tumors is entirely true, the surface epithelium of the ovary has a greater potentiality than that of any other adult tissue in the human body, and in this is exceeded only by that of fertilized ovum itself. Why disturb this firmly rooted and plausible belief as to the part it plays in the origin of ovarian tumors? We believe that acquired endometrial (including tubal) tissue in the ovary is of common occurrence. We desire to know its life history under normal and abnormal conditions, both on account of the scientific interest attached to it and in order that we may treat patients having these lesions more intelligently; we hope, somehow, to increase our knowledge of the conditions under which malignant changes may develop.

The evidence that ectopic endometrial tissue is often derived primarily from the implantation of bits of the uterine mucosa, which have been carried through the tubes by a back-flow of the menstrual blood into the peritoneal cavity and possibly occasionally from bits of the tubal mucosa is as definite as the evidence of the origin of any pathologic lesion in the human body. This has been fully considered in a previous publication.[4] I believe that menstrual blood escaping into the peritoneal cavity may possibly give rise to two very interesting groups of lesions. The first group includes those arising from the growth of the implanted bits of endometrial tissue. The second group consists of those arising from metaplasias of the peritoneal mesothelium and the surface epithelium of the ovary, due to their stimulation by the implantation or by some irritating substance in the menstrual blood. These metaplasias at times may resemble endometrial epithelium, and even gland-like

4. Sampson (Footnote 1, third reference).

inclusions may occur, thus simulating endometrial tissue and causing confusion in the diagnosis of the exact type of lesion present. If endometrial tissue, escaping through the tubes into the pelvis, should fall on suitable soil, it would grow and develop into "adenomas" of endometrial type, namely, Müllerian (endometrial) implants. The epithelial structure of the early implants is identical with that found in the uterine wall following its invasion by the musoca lining its cavity (compare Figs. 1, 2 and 4).

The nomenclature of these implants and the conditions arising from them is a difficult one to decide upon. As they apparently may arise from tubal as well as uterine mucosa, the term Müllerian would be inclusive and correct, but, unfortunately, it suggests an embryonic origin and is not as descriptive as endometrial. The term "adenoma" is hardly correct, since we cannot call the mucosal invasion of the uterine wall an adenoma. As their structure and also their reactions to menstruation, pregnancy [5] and the menopause are similar to those of the uterine mucosa and the conditions arising from the mucosal invasion of the uterine wall, I prefer at present to speak of them as endometrial implants and implantation endometrial invasions, hematomas, etc. The reaction of the tissues invaded may be slight, while in other instances there may be a marked increase in the connective tissue and smooth muscle about them, if the latter is present. Occasionally, a definite circumscribed leiomyoma may arise, enveloping the endometrial tissue, thus forming an endometrial leiomyoma (Fig. 4). The terms endometrial hematomas and endometrial cysts are descriptive and are correct, at least in the majority of the instances.

The primary endometrial implants are most often found in situations that would be reached readily by material escaping through the fimbriated opening of the tubes (Figs. 11 and 12). They occur most frequently in the posterior cul-de-sac, on the posterior surface of the uterus and on the lateral and under surfaces of the ovaries, but they may also be found at times on other structures in the pelvis. In some instances, they are present only on the peritoneum and in others only on the ovaries, but in the majority of the cases they are found on both the peritoneum and the ovaries. The peritoneal implants are usually slow-growing and insignificant, but they sometimes spread and become invasive. The ovarian implants are usually more active than the latter, thus suggesting that ovarian tissue is the most fertile soil. Frequently they invade the ovaries, developing into menstruating ovarian cysts which often perforate (Figs. 7, 28 and 33). The superficial ones are small, a few millimeters in diameter, while the deeper ones may reach a size of several centimeters (the largest one that I have observed was

5. Sampson (Footnote 1, second reference).

about 15 cm. in diameter). Any endometrial implant, wherever situated, may invade not only the tissues on which it primarily develops, but also adjacent structures with which it comes in contact. In its reaction to menstruation, epithelium apparently may be cast off and may give rise to other implants. The perforation of the ovarian endometrial cysts or hematomas, whether they are small or large, is apparently the chief source of these secondary implants. The ovary may be considered an

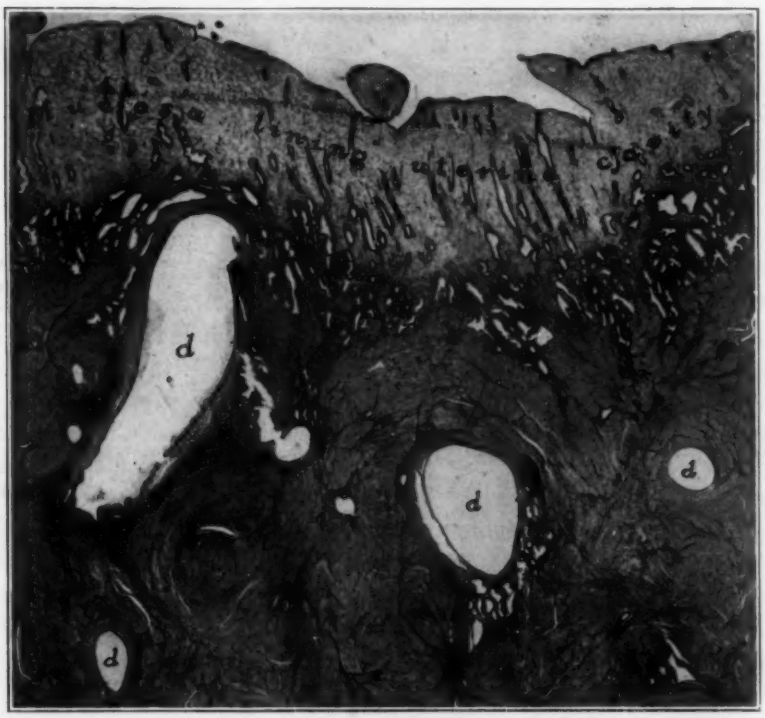

Fig. 1.—Invasion of the uterine wall by the mucosa lining its cavity; × 10. The patient, aged 53, complained of uterine bleeding. The operation was bilateral salpingo-oophorectomy, panhysterectomy and appendicectomy. The pathologic diagnosis was "adenomyoma" arising from the mucosal invasion of the uterine wall and "endometrial implants" in the cul-de-sac and right ovary (Figs. 18 and 23). The structures arising from the mucosal invasion of the uterine wall in this section consist of typical uterine mucosa and also isolated, normal-sized and dilated glands (*d*), some with abundant and others with little or no typical endometrial stroma about them. In this section, we recognize the fact that all of these structures are of endometrial origin. A few ciliated cells were found in the surface epithelium of the uterine mucosa, none in the normal-sized glands, but many in some of the dilated glands with flattened epithelium, although not in all of the latter. The various structures arising from the mucosal invasions of the uterine and tubal walls should be our standards in the study and diagnosis of endometrial implants. Only one or a few of the above mentioned endometrial structures may be present in a given implant.

incubator, hot-bed, redistributing focus, or even an intermediary host in the origin of secondary implants, and, in some instances, it may possibly impart greater activity (virulence) to the epithelium developing in it. The latter faculty, while apparently evident, is difficult to estimate, as there is such a great variation in the degree of invasiveness of the benign implants in different cases. Many are small and chiefly of histologic interest, while others may simulate malignancy in both their clinical and in their gross pathologic manifestations. In some

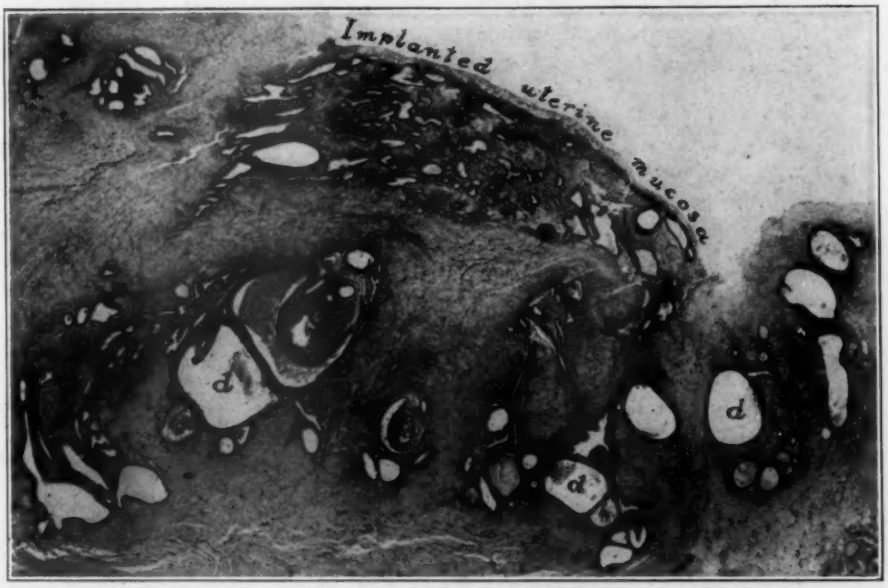

Fig. 2.—Invasion of the uterine wall by endometrial tissue implanted on its peritoneal surface; × 10. The patient, unmarried and aged 34, complained of pain in the lower left abdomen and severe dysmenorrhea. The operation was bilateral salpingo-oophorectomy, panhysterectomy and appendicectomy. The pathologic diagnosis was multiple leiomyomas (largest about 15 cm. in diameter), endometrial implants in both ovaries (Fig. 3), on the surface of the uterus, the leiomyomas, sigmoid and in the cul-de-sac. The histologic structure of the endometrial tissue on the surface of the uterus is similar to that of the uterine mucosa. The histologic structure of the glandular elements arising from the invasion of this tissue into the uterine wall is identical with that found in the invasion of the uterine wall by the mucosa lining its cavity shown in Figure 1. Cilia were not found on the surface epithelium of the implant or on the epithelium of the normal-sized glands, but were present in some of the dilated glands (*d*). The mucosa on the surface of the uterus is gradually "sinking into" the underlying uterine wall, and the tissues of the latter are growing over it. I believe that the endometrial tissue on the surface of the uterus and other pelvic structures in this case arose primarily from the implantation of bits of endometrial tissue escaping through the tubes during a retrograde menstrual flow.[4]

instances only primary implants are present, while in others there are both primary and secondary. When an ovarian hematoma is present with evidence of a previous perforation, and it is associated with adhesions and an endometrial invasion of the tissue involved in these adhesions, the endometrial tissue of the latter would seem in some instances to have arisen from epithelium carried with the contents of the hematoma escaping through the perforation (Figs. 29 and 33). The distribution of the implants, in these cases, is similar to that of those associated with malignant ovarian tumors, except that the latter are usually more extensive. I have never found a benign endometrial invasion of the omentum, which is so often involved in patients with peritoneal carcinosis.

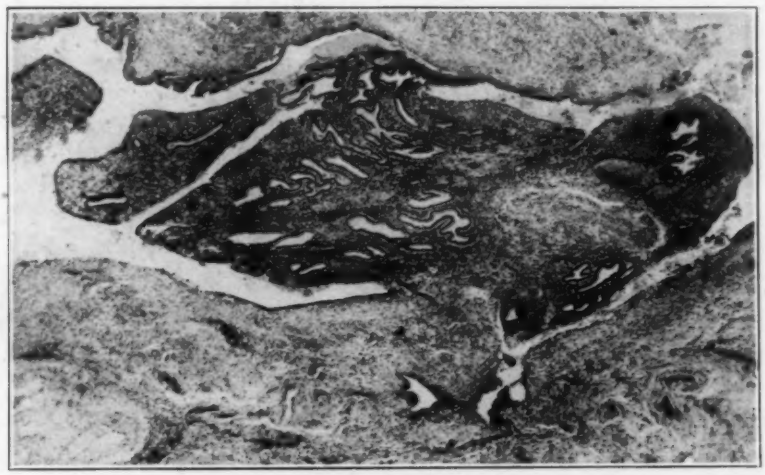

Fig. 3.—Endometrial tissue imbedded in the left ovary; × 25. Its histologic structure is similar to that of the endometrial implant embedded in the uterine wall shown in Figure 2 (the same case) and also to that of the uterine mucosa shown in Figure 1. On account of its typical histologic structure, it is easily recognized as endometrial tissue. Its origin has already been discussed in the legend of Figure 2. Ciliated epithelium was not found in any of its glands. Had it not been removed it would have been governed by the same natural laws as the mucosa lining the uterine cavity in its reaction to menstruation, pregnancy (if the patient had become pregnant) and the menopause. It might be slowly growing and persist as it is or give rise to an endometrial cyst several centimeters in diameter. As malignant changes occur in the mucosa lining the uterine cavity, so we infer that they may also arise in this misplaced endometrial tissue, giving rise to carcinoma of the ovary with the same histologic structure as that of the body of the uterus. Many ovarian cancers have the same histologic structure as cancer of the body of the uterus, and evidence of their origin (shown later) from endometrial tissue in the ovary may be found in some of these specimens.

Endometrial implants, wherever situated, usually react to menstruation, pregnancy and the menopause,[5] as does the mucosa lining the

Fig. 4.—Invasion of the uterine wall by endometrial tissue implanted on its peritoneal surface; two photomicrographs (× 10) of different portions of the same specimen. The patient, aged 56, complained of uterine bleeding. The operation was bilateral salpingo-oophorectomy, panhysterectomy and appendicectomy. The pathologic diagnosis was intramural and submucous leiomyomas and implantation endometrial invasion of both sides of the anterior uterine wall at the attachment of the anterior layer of the broad ligaments. Endometrial implants were not detected in the ovaries. It is more difficult to recognize these as endometrial implants than those shown in Figures 2 and 3, because typical uterine mucosa (a group of glands surrounded by endometrial stroma) is not present on the surface of the uterus. Histologically, normal-sized and slightly dilated uterine glands are present, some with and others without any endometrial stroma about them; in the area indicated by (*m*) of the lower photomicrograph typical uterine mucosa has developed from this invasion (compare Figs. 1 and 2). Circumscribed "adenoleiomyomas" (*Ad*) are present in both sections. A few ciliated cells were found in both sections, but only in dilated glands. The lower photomicrograph represents a later stage of the lesion present in the upper one. An apparent portal of invasion (arrow) has been almost obliterated by the endometrial tissue invading the uterine wall and by the tissues of the latter growing over and thus enveloping it. These two processes often make it difficult or at times impossible to detect the exact site of the original implantation. The gross appearance of these implants was similar to those shown in Figure 19.

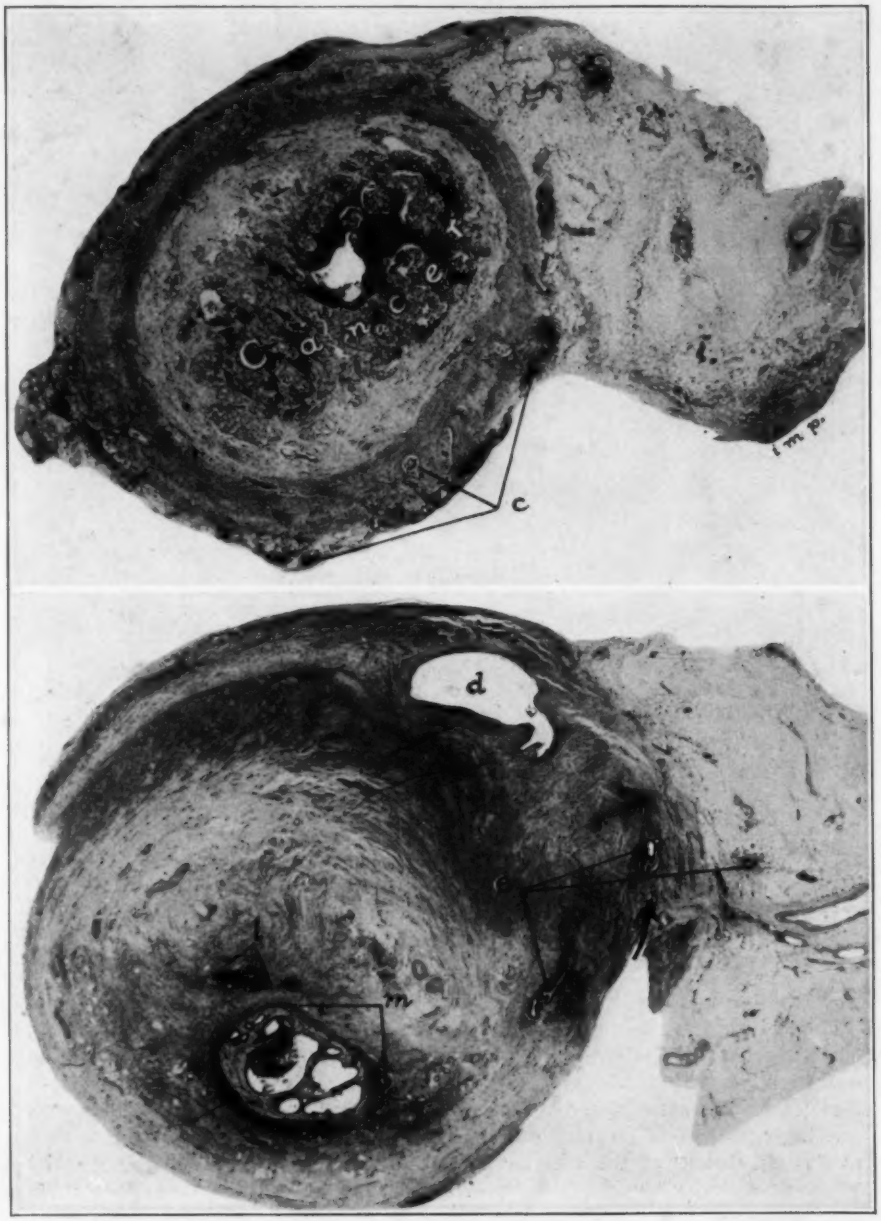

Fig. 5 (Case 4).—Cross section of two appendixes; × 10. The upper one has been invaded by carcinoma. Its lumen is obliterated, and the central portion of the appendix is almost completely replaced by cancer, which has invaded the appendix from its peritoneal surface (*c*). The carcinoma apparently arose in endometrial tissue in the ovary. The lower appendix (section taken near the tip) has been invaded by benign endometrial tissue from its peritoneal surface at the attachment of the mesoappendix (arrow). Normal-sized (*e*) and dilated glands (*d*) have arisen from this invasion as well as a mass of uterine mucosa (*m*), which is analogous to the cancer in the center of the upper photomicrograph. Ciliated epithelium was present in some of the glands. The lumen (*l*) of the appendix is obliterated in this portion of the appendix. The patient, aged 37, married but never pregnant, had multiple uterine leiomyomas and endometrial tissue in both ovaries, the posterior uterine wall, the cul-de-sac, the left round ligament, the terminal loop of the ileum and the appendix. The endometrial tissue invading the appendix may have been derived from other ectopic endometrial lesions in the pelvis or from tissue escaping through the tubes with a back-flow of menstrual blood into the peritoneal cavity. The malignant invasion of the appendix is a well-recognized implantation lesion. I believe the other is a benign implantation lesion, and that neither can be attributed to a metaplasia of the serosa of the appendix.

uterine cavity. Thus, they are governed by the same natural laws as the latter, and one would infer that they would be liable to similar pathologic changes, including malignancy, if exposed to the exciting cause. One would infer that cancer might develop in any implant or structure resulting from it, wherever situated, and especially in those in the ovary, since they are apparently the most active. One would also infer that cancer might be more likely to develop in implants of uterine origin, rather than in those of tubal origin, because the uterine mucosa is more active in its response to normal stimuli, such as menstruation and pregnancy, and is more frequently the seat of cancer.

The development of cancer in these endometrial implants, with special reference to those of the ovary, is the subject of this paper.

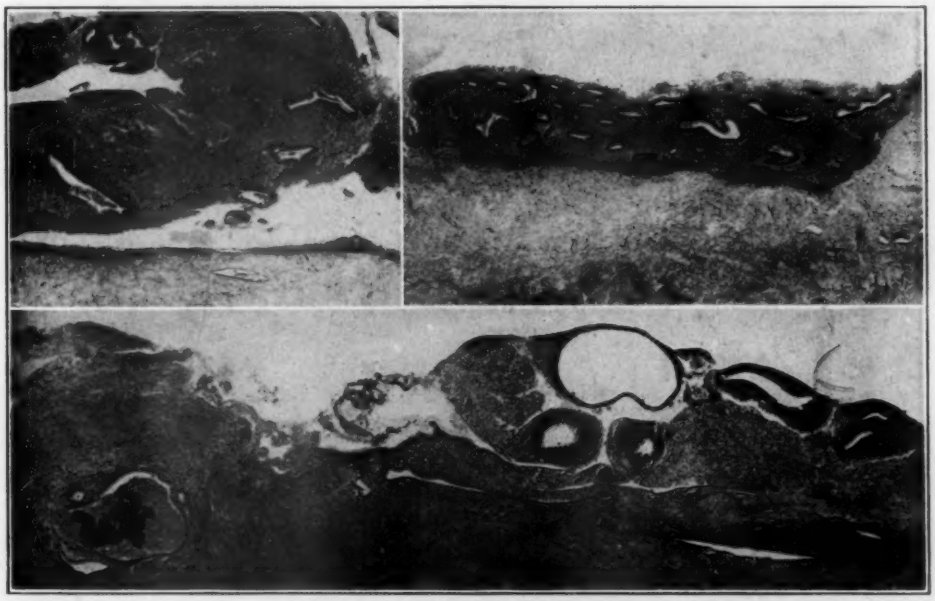

Fig. 6.—Variations in the type of endometrial tissue implanted on the ovary; × 10. The two upper specimens are from the same patient, aged 47, with endometrial implants on the under surface of each ovary and on the posterior surface of the uterus. The one on the left is attached to the surface of the ovary and has not invaded it. The one on the right is embedded in the ovary. Both have the typical histologic structure of uterine mucosa. The lower specimen is from a patient, aged 37, with multiple leiomyomas and endometrial implants on both ovaries, a resulting endometrial hematoma of the left (Fig. 7) and implants on the posterior uterine wall, sigmoid and ileum. The condition shown in this photomicrograph is more difficult to recognize as endometrial tissue, but a careful study of the section (Fig. 10) shows that the endometrial elements in this section are histologically identical with some of those found in the mucosal invasion of the uterine wall shown in Figure 1. This is the more frequent type of lesion found in endometrial implantation irrespective of the organ or tissue involved. Its variations will be more fully presented, as they are important to recognize.

EVIDENCE SUGGESTING THAT CARCINOMA MAY ARISE IN ENDOMETRIAL TISSUE IN THE OVARY

The evidence in favor of malignant changes in endometrial tissue in the ovary, as the source of certain ovarian cancers, may be summarized thus:

1. Endometrial tissue in the ovary is of common occurrence. During the year from May 1, 1922, to May 1, 1923 (representing a little less than eleven operative months) I encountered sixty-four instances of endometrial implantation in 296 abdominal operations for pelvic

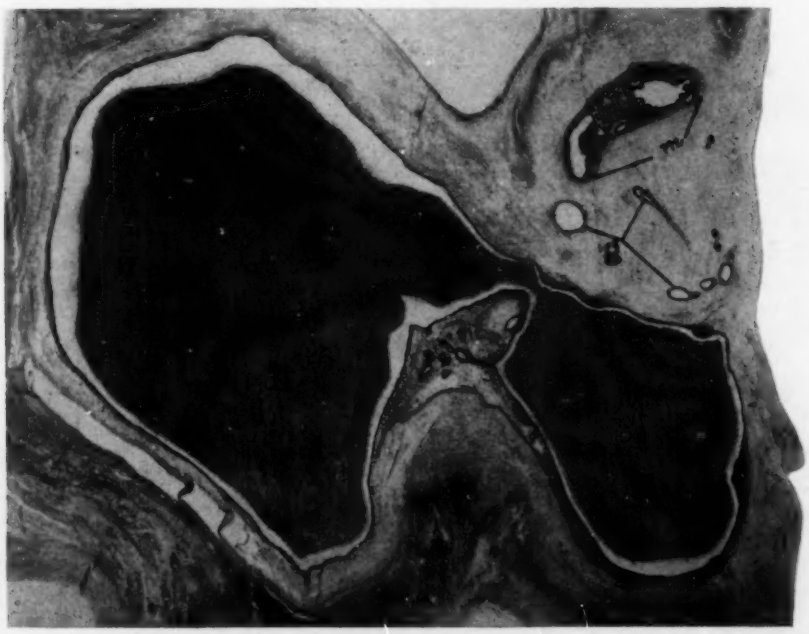

Fig. 7.—Endometrial invasion of the ovary from endometrial tissue implanted on its surface, giving rise to the glandular elements (*g*) found in the mucosal invasion of the uterine wall (Fig. 1), and also to a mass of typical uterine mucosa (*m*) and an endometrial cyst or hematoma; retouched photomicrograph (×7). These hematomas may reach a large size (the largest I have encountered was 15 cm. in diameter). This is from the same ovary shown in the lower specimen (Fig. 6) and only a few millimeters from the latter (Fig. 10).

disease. In forty-five of these patients, one or both ovaries were involved. During the year from May 1, 1923, to May 1, 1924 (representing a little less than eleven operative months) ninety-eight instances of endometrial implantation were encountered in 332 operations similar to the above. In sixty-four of these patients, one or both ovaries were

involved. Certain questions naturally arise. Why should not this tissue become malignant at times, and if so, what forms would it be likely to assume?

2. I believe that this ectopic endometrial tissue might become malignant because in its response to menstruation, pregnancy and the menopause it is governed by the same natural laws as the mucosa lining the uterine cavity, and the latter becomes malignant; the possibility is increased because of the additional fact that it is misplaced.

3. One of the most striking features of benign endometrial ovarian cysts with their associated peritoneal implants is their close resemblance to certain malignant ovarian tumors (including ovarian cysts) and their associated peritoneal implants. Many adenocarcinomas of the ovary have the same histologic structure as adenocarcinoma of the body of the uterus.

4. Malignant ovarian tumors are frequently bilateral, as are also benign endometrial growths in the ovary.

5. Bits of cancer from cancer of the body of the uterus may escape through the tubes causing implants on the peritoneum and the surface of the ovaries, and these implants may develop into ovarian carcinomas.[4]

6. The age incidence of certain types of ovarian cancers corresponds with that of cancer of the body of the uterus (endometrial carcinoma).

7. The first instance of endometrial tissue in the ovary reported, according to Russell,[6] was associated with adenocarcinoma of the opposite ovary.

8. Ovarian papilloma, which frequently becomes cancerous, has been reported arising from the fimbriae of the tube.[7]

9. This acquired endometrial tissue in the ovary, which is of common occurrence, offers in part the most satisfactory explanation of the frequency of ovarian carcinoma as compared with that of the testis, both organs having a common embryonic origin.

The foregoing evidence is circumstantial, but nevertheless valuable. However, it is not conclusive. Not only is there needed the actual demonstration of both cancer and benign endometrial tissue in the same ovary, the two bearing the same histologic relation to each other that cancer of the body of the uterus bears to the nonmalignant portions of the endometrium of that uterus, in order to indicate the possible origin of ovarian carcinoma in this tissue, but, to make it conclusive, it must be shown that the cancer arose in this tissue and that it is not invading

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6. Russell, W. W.: Aberrant Portions of the Müllerian Duct Found in the Ovary, Bull. Johns Hopkins Hospital **10**:8-10, 1899.

7. An exhaustive discussion of the surface epithelium of the ovary, and the origin of ovarian tumors from it is given by Williams, J. W.: Papillomatous Tumors of the Ovary, Reports John Hopkins Hospital **3**:1-84, 1894.

it from some other source. This is much more difficult to prove in the ovary than in the uterus because there are other structures in the ovary which are generally accepted as the possible source of cancer.

As a foundation for the study of this problem, one must be able to recognize the gross appearance and the histologic structure of the various lesions resulting from endometrial implantation, not only in the ovary, but also in the other pelvic structures. While it is true that in some cases they may be present only in the ovary or ovaries, or only in other pelvic structures, they often occur in both. If benign peritoneal implants and the lesions resulting from them are found in a

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EXPLANATION OF PLATE I

Fig. 8 (Case 6).—A recent carcinomatous implant on the posterior surface of the uterus (Fig. 66); from a colored photomicrograph with a magnification of 60 diameters. The reaction of the host against the implantation is well shown. I believe that the condition shown in this illustration is an implant arising from malignant cells escaping into the peritoneal cavity from the primary ovarian cancer. Histologically, it is an adenocarcinoma of the same histologic structure as that in the ovary and the cancer in the right ovary had extended beyond that organ (Fig. 67). The ovarian carcinoma possibly arose in endometrial tissue in that organ. Comparison should be made with Figure 9.

Fig. 9.—A recent benign endometrial implant on the posterior surface of the uterus of a patient, aged 37; from a colored photomicrograph with a magnification of 60 diameters. It consists of glands and tissue about them histologically identical with that of the uterine mucosa. The reaction of the host is similar to that shown in the preceding illustration, and the general histologic structure of the two lesions is the same. What is the source of this implant? A similar lesion was present on the surface of the left ovary. It may have arisen from this as the implant in the preceding illustration arose from malignant (possibly endometrial) tissue in the ovary, or it may have arisen from endometrial tissue escaping through the tubes with a back-flow of menstrual blood. We recognize the lesion in Figure 8 as an implantation. Should not this one also be considered an implantation, rather than a localized metaplasia of the serosa of the uterus?

Fig. 10.—A benign endometrial implant on the surface of the ovary, from the same section shown in Figure 6, but a higher magnification; from a colored photomicrograph with a magnification of 60 diameters. The general histologic structure of the lesion is that of an implantation. The glands have the histologic structure of uterine glands with tissue about them resembling endometrial stroma, and evidence of old and recent hemorrhage into the same. The dilated gland (*d*) with a flattened epithelial lining has the same structure as the dilated glands shown in Figure 1. The space (*s*) is lined with the surface epithelium of the ovary. I believe this is an endometrial implant not only on account of its histologic structure, but because similar lesions were found on other pelvic structures and the ovary (Fig. 7) had been invaded by this tissue, giving rise to endometrial tissue in that organ and a typical ovarian endometrial hematoma.

PLATE I

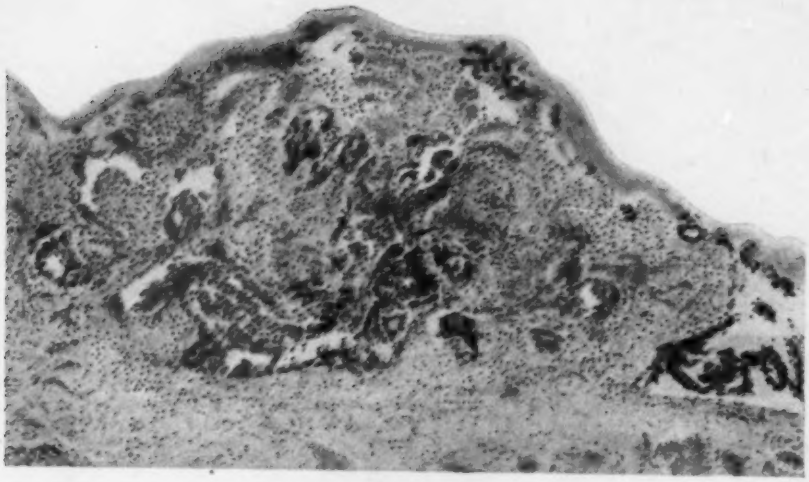

Fig. 8

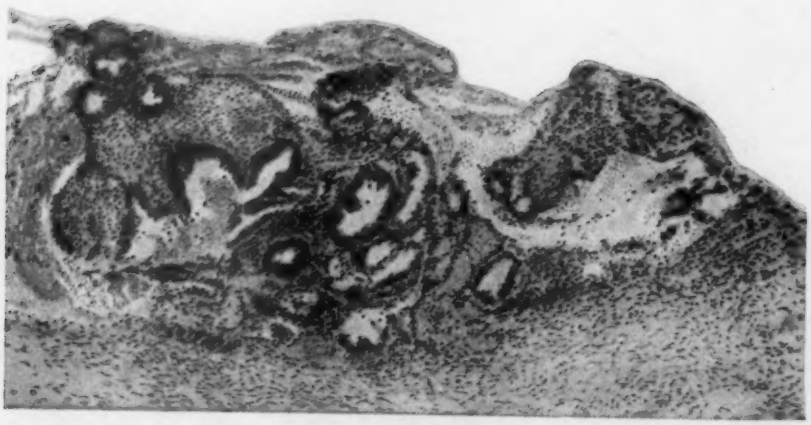

Fig. 9

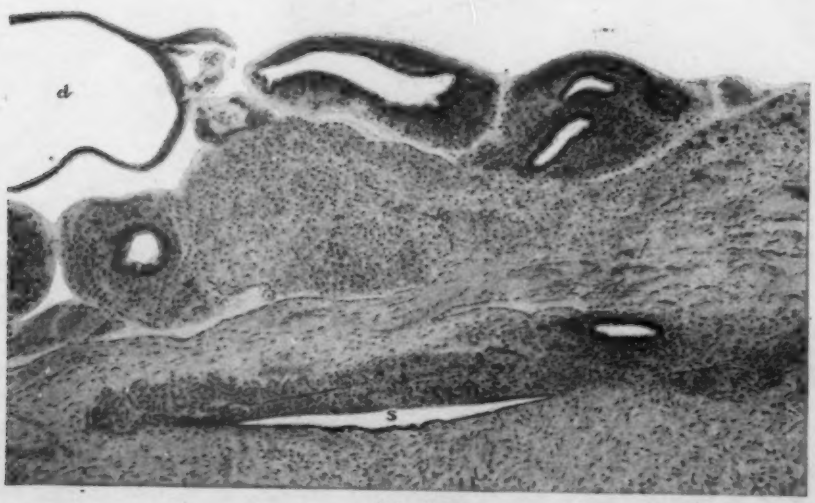

Fig. 10

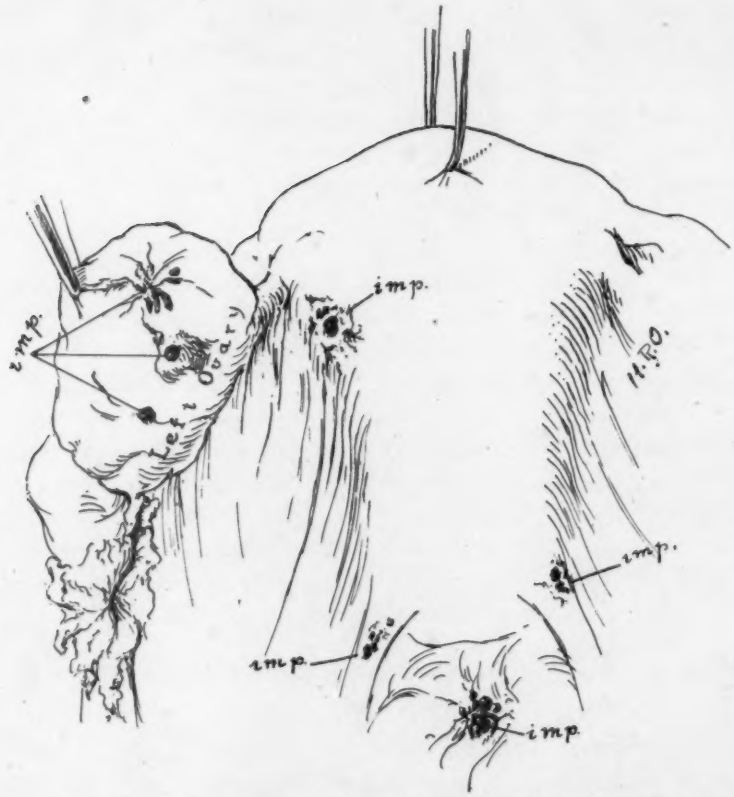

Fig. 11.—Endometrial implants (*imp.*) on the lateral and under surfaces of the left ovary, on the posterior surface of the uterus, both uterosacral ligaments and on the anterior surface of the rectum, as found at operation (natural size). The patient, aged 23, complained of dysmenorrhea and pain in the right lower abdomen. She was married, but had not had any children. The uterus was retroflexed. The operation was dilatation of the cervix, appendicectomy, left salpingo-oophorectomy, excision of peritoneal implants and suspension of the uterus. The gross appearance of all of the implants was similar. Their histologic structure (Fig. 21) in each instance consisted of some of the endometrial elements found in the mucosal invasion of the uterine wall (Fig. 1) and in addition hemorrhage (inferred as menstrual) into their tissues. Both tubes were patent. The histologic structure of the implants, their gross appearance, their distribution, the knowledge that menstrual blood sometimes escapes through the tubes and that bits of endometrial tissue transplanted in human beings may grow, indicates both the probable nature of these lesions and their probable origin (Figs. 13, 16 and 21).

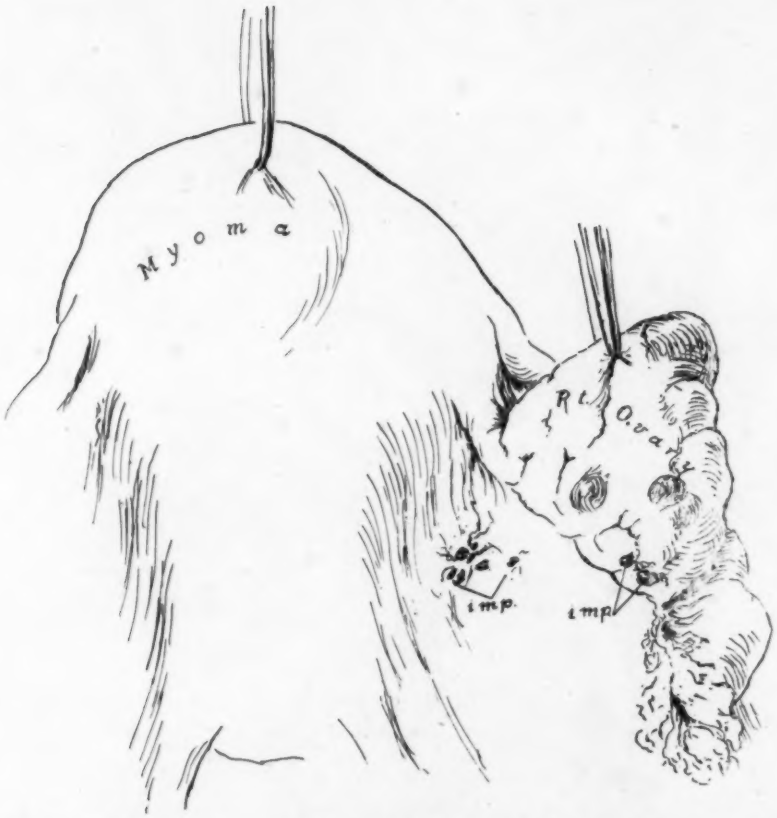

Fig. 12.—Endometrial implants (*imp.*)) on the lateral surface of the right ovary and on the posterior surface of the right broad ligament near its attachment to the uterus; intramural leiomyoma of the uterus (natural size). The patient, aged 29, complained of dysmenorrhea and profuse menstruation. She had been married seven years and had never been pregnant. The uterus was slightly enlarged because of an intramural leiomyoma, and was retroflexed. The operation was appendicectomy, right salpingo-oophorectomy and supravaginal hysterectomy. The gross appearance of the implants was similar to those shown in Figure 11 (also Figs. 14 and 17). Their histologic structure may be seen in Figures 15 and 22. If the patient had not been operated on, other implantations might have occurred; these might have remained small and insignificant. On the other hand, ovarian hematomas might have developed, similar to those shown in Figures 28 and 33. If cancer should arise in the endometrial tissue of an ovary similar to this one, malignant peritoneal implantations would occur early, and, as the amount of ectopic endometrial tissue is small, it might be difficult or impossible to detect any benign endometrial tissue at the time of the operation or find it in the specimen removed, as suggested in Cases 6 and 7.

patient with ovarian carcinoma, there probably has been, or still is, in one or both ovaries benign endometrial tissue in some portion of which the carcinoma may have developed.

It is of the greatest importance to be able to recognize these benign endometrial lesions in women after the menopause, because carcinoma of the body of the uterus and certain types of ovarian carcinoma develop most frequently in elderly women. The opportunity to study these benign endometrial lesions in elderly women is not afforded as often as it is in women before the menopause. These lesions are most often found during operations for uterine leiomyomas, retroflexion of the uterus and for the relief of conditions resulting from endometrial implantation. The most marked symptoms of the latter arise from the reaction of these lesions to menstruation. They respond at the menopause in the same manner as the uterine mucosa, and therefore rarely cause sufficient discomfort in elderly women to warrant operative interference. Leiomyomas of the uterus also rarely require operative treatment after the menopause. Only sixteen of the 162 patients referred to in this communication were over 50 years of age. Nevertheless, sufficient material has been obtained to furnish a clear conception of their normal life history. The usual changes caused by the menopause in all misplaced endometrial tissue is identical with that brought about in the mucosa lining the uterine cavity.

THE DIAGNOSIS OF BENIGN ENDOMETRIAL IMPLANTS AND THE CONDITIONS RESULTING FROM THEM

These implants can usually be readily detected at operation by those familiar with them. The pigmentation due to the retention of menstrual blood when present and the characteristic reaction of the tissues invaded present a picture rarely simulated by any other condition (Figs. 13, 14, 16, 17, 18 and 19). It is often more difficult to detect them in the specimen removed, because superficial hematomas are apt to be ruptured during the operation, and the lesions may be so mutilated otherwise as to be easily overlooked by the pathologist.

Bits of endometrial tissue becoming implanted on the surface of any pelvic structure develop either into normal-sized or dilated glands with little or no endometrial stroma about them (Figs. 4 and 10) or into a mass of typical uterine mucosa (a group of glands surrounded by endometrial stroma) (Figs. 2, 3 and 6). Possibly, in the first instance, the tissue causing the implant consisted of epithelium with little or no stroma, and, in the second, epithelium plus a definite amount of stroma was carried with the menstrual blood escaping through the tubes into the peritoneal cavity. The tissue beneath the implant reacts to it as to a foreign body or a malignant implant (compare Figs. 8 and

EXPLANATION OF PLATE II

Fig. 13.—Left tube and ovary (natural size), the latter turned upward, exposing the endometrial implants on its lateral and under surfaces (Fig. 11). The pigmentation is due to hemorrhage (which we infer is menstrual) into the tissues of the implant. The histologic structure of the implants is identical with that shown in Figure 15, except that the implants are embedded more deeply in the tissues of the ovary.

Fig. 14.—Right tube and ovary (natural size), the latter turned upward exposing the endometrial implants on its lateral surface, also shown in Figure 12. The implants are more superficial than those shown in Figure 13. The histologic structure of the larger implant may be seen in Figure 15.

Fig. 15.—The larger endometrial implant on the ovary shown in Figures 12 and 14; from a colored photomicrograph with a magnification of 60 diameters. It has the histologic structure of a dilated uterine gland (compare with Figures 1, 2, 10 and 22). Its epithelium is low, cuboidal and columnar, and an occasional ciliated cell is present. Its epithelial lining does not resemble the surface epithelium of the ovary near it, although one might infer that it had risen from it. The tissue about its base suggests endometrial stroma. The vessels in this tissue are dilated, and there is extravasation of blood in this tissue and also into the lumen of the dilated gland. This hemorrhage might be considered mentrual, as might also the hemorrhage in the peritoneal implant. While I am unable to exclude its origin from the surface epithelium of the ovary, I believe that it is more apt to be an endometrial implant not only on account of its histologic structure, but also because of its situation (endometrial implantations usually occur on the lateral and under surface of the ovary), its gross appearance and the presence of a peritoneal implant with the same gross appearance and histologic structure (Figs. 12, 14, 17 and 22).

Fig. 16.—Endometrial implant (natural size) on the anterior surface of the rectum (shown in Fig. 11). Its histologic structure may be seen in Figure 21.

Fig. 17.—Endometrial implant (natural size) on the posterior surface of the right broad ligament (shown in Fig. 12). Its histologic structure may be seen in Figure 22.

Fig. 18.—"Endometrial" implant (natural size) in the cul-de-sac, referred to in Figure 1. It possibly represents the latter phase of a dormant peritoneal implant, as shown by the increase in the scar tissue in it (compare Figs. 16 and 17). Its histologic structure may be seen in Figure 23.

Fig. 19.—Two peritoneal endometrial implants (natural size), both on the anterior surface of the uterus about the attachment of the round ligaments. Similar implants were present in the anterior and posterior cul-de-sac, but none in the ovaries. They have ceased to react to menstruation (the patient was aged 51), and the characteristic pigmentation due to the retention of menstrual blood has disappeared from the lower implant. The histologic structure of these implants was similar to that of those shown in Figure 4.

Fig. 20.—Cancerous peritoneal implants (natural size), taken from a patient with a malignant ovarian cyst and evidence of a recent perforation (Case 4). The cancerous ovarian cyst had apparently arisen from malignant changes in endometrial tissue in the ovary. These implants differ in their gross appearance from the benign endometrial implants shown in the preceding illustrations and should not be confused readily with them.

PLATE II

Fig. 13

Fig. 14

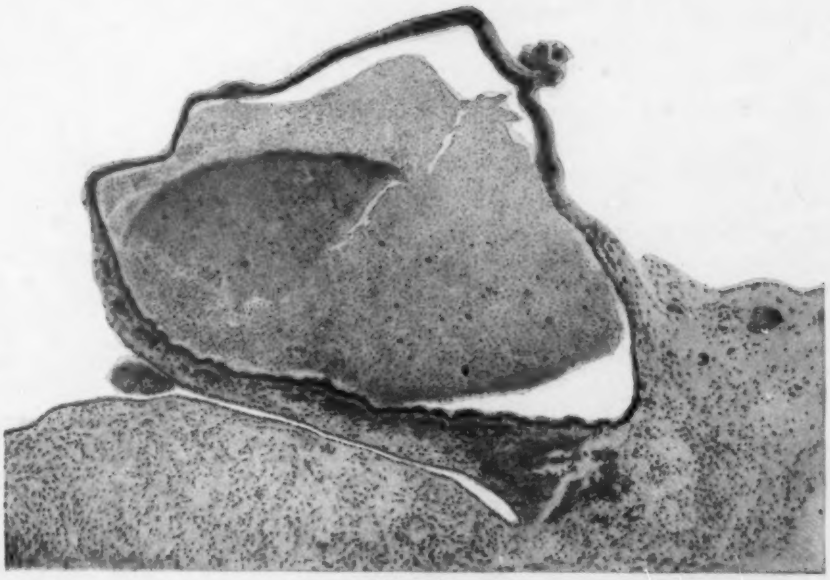

Fig. 15

Fig. 16

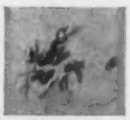

Fig. 17

Fig. 19

Fig. 20

Fig. 18

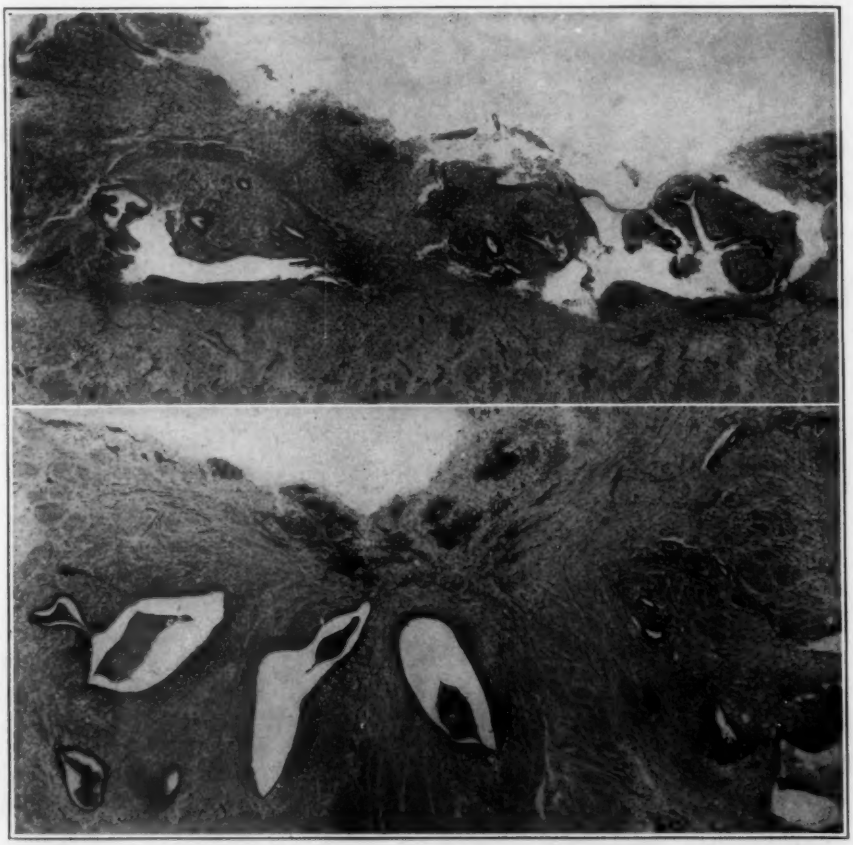

Fig. 21.—Sections of the implant on the rectum (Figs. 11 and 16) and of the one on the right uterosacral ligament (Fig. 11); × 25. The histologic structure of the ovarian implants was discussed in the legend of Figure 13. The rectal implant (upper photomicrograph) shows quite typical uterine mucosa on the surface of the rectum, but without any ciliated epithelium. Hemorrhage into this tissue gave rise to its gross appearance (shown in Fig. 16). The uterosacral implant (lower photomicrograph) possibly represents a later stage of the preceding, i. e., it is an older implant, the endometrial tissue having invaded its host, with the portal of entry not evident in this section, or it may have been obliterated (compare Fig 4). The glands are accompanied by very little endometrial stroma, some without any; hemorrhage was present in the tissue about some of the glands and in their lumina. A few ciliated cells were found in its dilated glands. The diagnosis of endometrial implants in this case (Figs. 11, 13 and 16) is based on their distribution, gross appearance and histologic structure, the latter corresponding to some of the glandular elements found in the invasion of the uterine wall by its mucosa (Fig. 1).

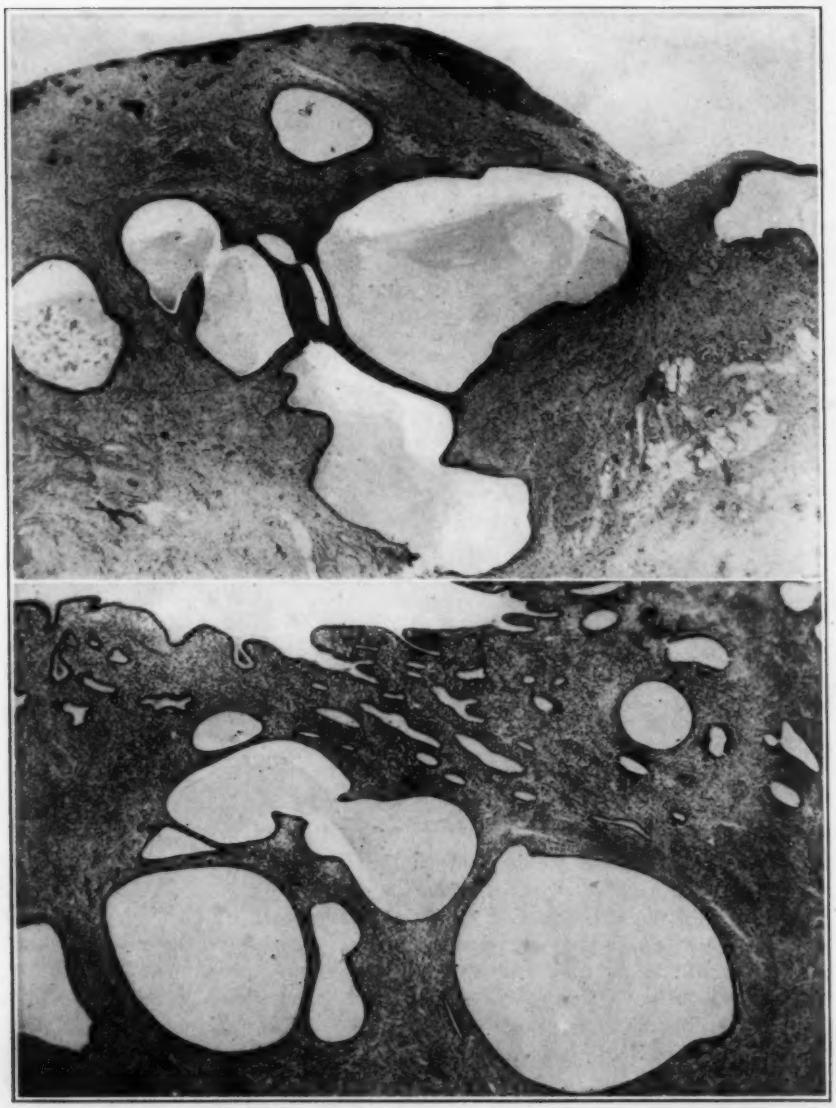

Fig. 22.—Two photomicrographs (×25), the upper one of a section of the peritoneal implant shown in Figures 12 and 17, and the lower of a portion of the mucosa of the uterus of the same case, the latter showing glandular dilatation. The dilated glands in both sections, as well as the one shown in Figure 15 (from the implant on the ovary), have the same histologic structure, including the presence of ciliated epithelium. I believe that all these dilated glands in the three different situations could be of endometrial origin.

9). The degree of reaction apparently varies greatly in different cases, and also varies with the age of the implantation. The implant may remain superficial, becoming localized or spreading over the surface of its host. In other instances, it invades the underlying tissue and the adjacent structures, which may become adherent to it. The invasion

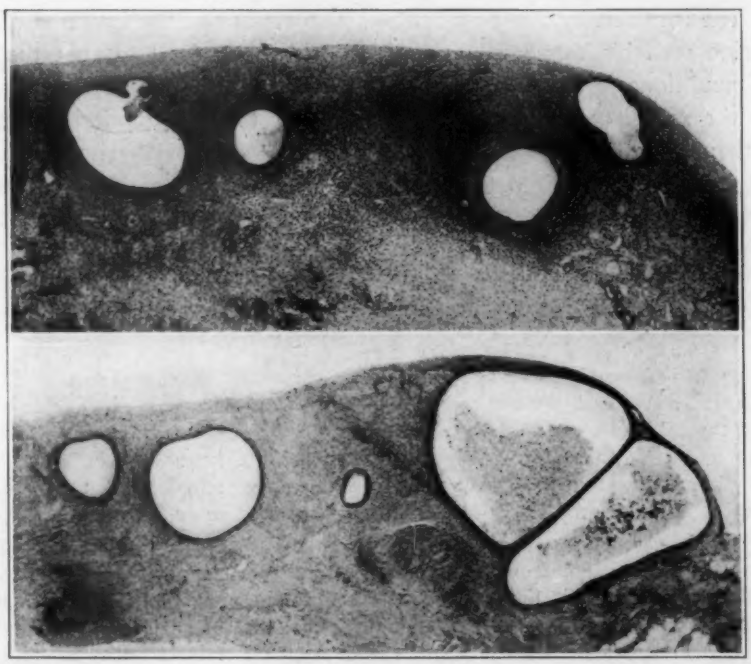

Fig. 23.—Two photomicrographs (×25), the upper one of a section of a portion of the right ovary and the lower of a peritoneal implant in the cul-de-sac (Fig. 18), from the same case shown in Figure 1. The dilated glands in the ovary are lined by epithelium, low cuboidal to columnar, and some of them are ciliated. There is no tissue about them resembling endometrial stroma, or any reaction suggesting menstruation. Did these glands arise from uterine or tubal epithelium implanted on the surface of the ovary, or from the true surface epithelium of that organ? I believe that it is impossible to state their origin in this case. Their structure is similar to that of some of the dilated glands shown in Figures 1 and 2 and also with the structure of those in the photomicrograph of the peritoneal implant (lower photomicrograph). In the latter, there is a small amount of tissue about some of the glands resembling endometrial stroma, and evidence of hemorrhage into this and into the lumina of some of the glands. This specimen is an example of an instance in which it is impossible (with our present knowledge) to make an exact diagnosis. I believe that the glands on the ovary could be of implantation origin. The conditions shown in Figures 11 to 23 inclusive (excepting Fig. 20), have been purposely chosen as representing lesions in which an exact diagnosis is difficult. I believe that all of them could have arisen from endometrial implantations as indicated in the legends.

by this endometrial tissue is similar to the invasion of malignant implantation and is brought about by two factors, (1) the actual invasion of the host by the endometrial tissue and (2) the growth of the tissue of the host about it and enveloping it. The latter process may make it difficult or impossible to detect the exact site of the original implantation (Fig. 4).

The invasion by these implants is also similar to the invasion of the uterine wall by its mucosa (compare Figs. 1 and 2), except that, in the conditions resulting from endometrial implantation there is no outlet for the menstrual blood (should they react to menstruation, as many of them do); this blood is retained in the tissues of the implant or it escapes by rupture into the peritoneal cavity. The retention of menstrual blood gives rise to endometrial hematomas, which reach their

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EXPLANATION OF PLATE III

Fig. 24.—A portion of the wall of the ovarian hematoma shown in Figure 28; from a colored photomicrograph with a magnification of 25 diameters. It is typical endometrial tissue, including glands, with an extensive stromal hemorrhage. Some of the epithelial lining of the hematoma has been cast off by blood escaping into its cavity. An interesting metaplasia of the epithelial lining of other portions of the hematoma may be seen in Figure 29.

Fig. 25.—Another portion of the wall of the endometrial ovarian hematoma shown in Figure 28; from a colored photomicrograph with a magnification of 60 diameters. It is lined by columnar epithelium with evidence of a small subepithelial hematoma. The histologic structure shown here is frequently found in endometrial hematomas of the ovary, and it is in such an epithelial lining that cancer may arise (Fig. 65).

Fig. 26.—Variations in the histologic structure of the lining of the large endometrial cyts or hematoma (Fig. 33); from colored photomicrographs with a magnification of 60 diameters. The one at the left presents a later stage of the lining shown in Figure 25, as indicated by the phagocytic cells in its stroma, while the one at the right shows the characteristic lining consisting of phagocytic cells and blood pigment, resulting from hemorrhage into the tissues of the former, with casting off of the overlying epithelium and the subsequent reaction of the underlying stroma. Both conditions are frequently found in benign, and sometimes in malignant endometrial cysts (Fig. 55).

Fig. 27 (Case 4).—A portion of the wall of a small loculus that communicated with the cavity of a large carcinomatous ovarian cyst (Fig. 52); from a colored photomicrograph with a magnification of 130 diameters. The greater portion of this loculus was lined by benign endometrial tissue (Figs 53 and 54); this condition is also indicated in the left half of this photomicrograph. Carcinoma is shown in the right half. At the junction of the benign and malignant epithelial lining, cancer can be seen replacing or arising from the benign epithelium, a condition that is often found in cancer of the body of the uterus.

PLATE III

Fig. 24

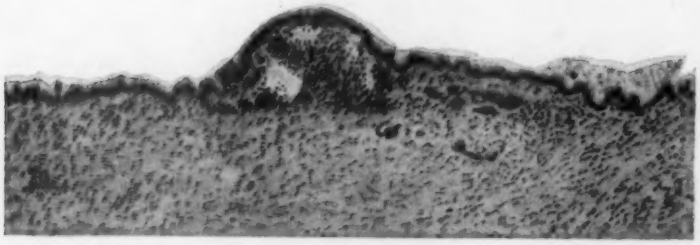

Fig. 25

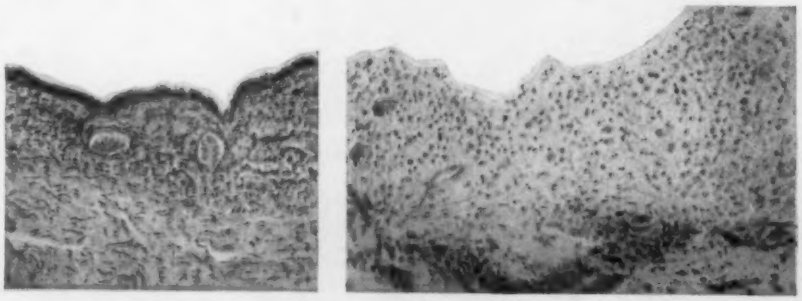

Fig. 26

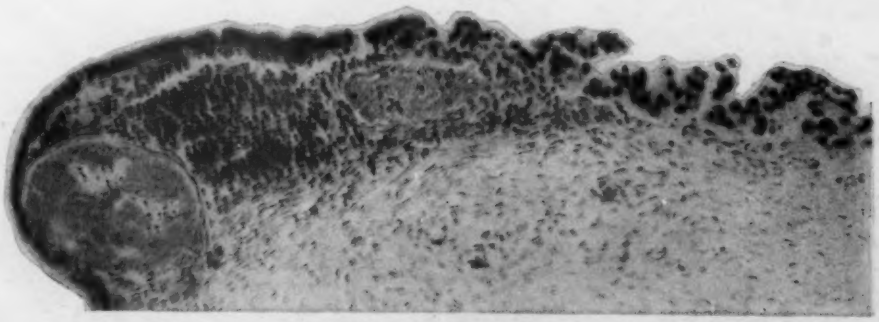

Fig. 27

greatest development in the ovary. Our standard for comparison (diagnosis) in the study of conditions resulting from endometrial implantation must be based on the conditions resulting from the mucosal invasion of the uterine and tubal walls. This must be modified by the kind of tissue invaded and by the retention of menstrual blood. A study of the mucosal invasion of the uterine wall demonstrates that the endometrial elements represented in this invasion often show a varied structure in different areas of the same section. In some areas, typical uterine mucosa may be present, in other areas dilated glands with flattening of the epithelial lining, and again normal-sized or dilated glands with little or possibly no endometrial stroma about them (Fig. 1) may be found. In these sections, we recognize that the glands with little or no stroma about them are of endometrial origin. In some endometrial implants, typical uterine mucosa is present on the surface of the host and there may be associated with it the glandular invasion of the underlying tissue, similar in its histologic detail to that of the mucosal invasion of the uterine wall just described (compare Figs. 1 and 2). In these instances likewise, we do not hesitate to state that the glands not accompanied by stroma, which have invaded the tissues of the host from the endometrial tissue on its surface, are also of endometrial origin. In the majority of implants, typical uterine mucosa is absent, but normal-sized or dilated glands are often present, with little and sometimes with no endometrial stroma about them (Fig. 4). If typical uterine mucosa should develop in the tissues invaded by these glands, we could recognize the superficial glands to be of endometrial origin. If there were no typical uterine mucosa in the section, the diagnosis would be more difficult. Further study of the sections of mucosal invasion of the uterine wall gives some assistance. The detection of ciliated epithelium in normal uterine mucosa is sometimes easy and sometimes difficult, while ciliated cells are easily found in sections from the tubes accompanying the uterus whose mucosa has been examined. The search for ciliated epithelium in sections of uterine mucosa with dilated glands, and also in the mucosal invasion of the uterine wall, indicates that they are often more readily found in the dilated glands with flattened epithelium than in the normal-sized glands (Figs. 1 and 22). The same is true in cases of endometrial implantations (Figs. 2 and 22). It may be impossible always to find ciliated cells in a section of typical endometrial tissue, wherever it is situated, but if we can find them in misplaced endometrium-like tissue, it may be of assistance in the diagnosis. The dilated glands offer the best field for detecting them, and dilatation of the glands is the very condition that is often the most difficult to diagnose. I have purposely discussed these dilated uterine glands and the detection of ciliated epithelium in them, because the former play an important part in the specimens of malignant ovarian

tumors which I wish to present. If cilia are found, it sometimes assists in the diagnosis, but if they are not found, it does not exclude the possibility of the gland under observation being of endometrial origin. The histologic study of the mucosa of the uterus accompanying the specimen may fail to show any ciliated epithelium.

I have carefully studied the changes in the mesothelium of the peritoneum and in the surface epithelium of the ovary brought about to their reaction (1) to the hemorrhage of tubal pregnancy, (2) to pelvic peritonitis and (3) to benign and malignant epithelial implantations. I appreciate how closely these changes may sometimes simulate endometrial epithelium and also the fact that gland-like inclusions of the peritoneal mesothelium and of the surface epithelium of the ovaries may result, and that sometimes it may be difficult or even impossible to determine whether the given tissue under observation arose from the reaction just described or from the implantation of tissue escaping through the tubes. In some instances, gland-like structures from both sources are present. Menstrual blood may be an ideal irritant for the stimulation of the peritoneal mesothelium and the surface epithelium of the ovary. I have not yet been convinced that typical endometrial mucosa with its characteristic stroma can arise in these metaplasias.

Gland-like structures in the ovary, without endometrial stroma about them, apparently having arisen from the downward growth of the true surface epithelium of the ovary, are of not infrequent occurrence. Their epithelial lining may be low, cuboidal or columnar, and cilia are sometimes present. It is generally accepted that these gland-like structures arise from the true surface epithelium of the ovary and that the latter may sometimes be ciliated.[7] Some of these glands may have developed before birth and others after birth. It is believed that uterine and tubal epithelium often becomes implanted on the surface of the ovary or on any other pelvic structure and, by invasion, gives rise in the tissues of its host to glands, which are not necessarily accompanied by endometrial stroma. It is also known that the epithelium of these glands arising from implanted uterine and tubal epithelium may be ciliated just as the normal uterine and tubal mucosa may be ciliated. The absence of cilia in a given gland, whether it is situated in the mucosa lining the uterine cavity or in ectopic endometrium-like tissue, does not exclude that gland from having been derived from uterine or tubal epithelium. Does the presence of cilia indicate that it was not derived from the true surface epithelium of the ovary, but did arise from uterine or tubal epithelium implanted on its surface? I have never been able to find cilia on the peritoneal mesothelium, and in only one instance have I found them on the surface epithelium of the ovary. In some specimens, I have been able to satisfy myself that ciliated glands similar to those just described arose from implanted epithelium, and in other

instances I have been unable to demonstrate this origin and have assumed that they might have arisen from the surface epithelium of the ovary either before or after birth. My present reaction is that these glands in the ovary with ciliated epithelium are sometimes acquired after birth from the implantation of uterine or tubal epithelium on the surface of the ovary. Certainly, when they are associated with lesions of the same gross appearance and the same histologic structure situated on the surface of other pelvic structures, which could easily have been contaminated by menstrual blood escaping through the tubes, the

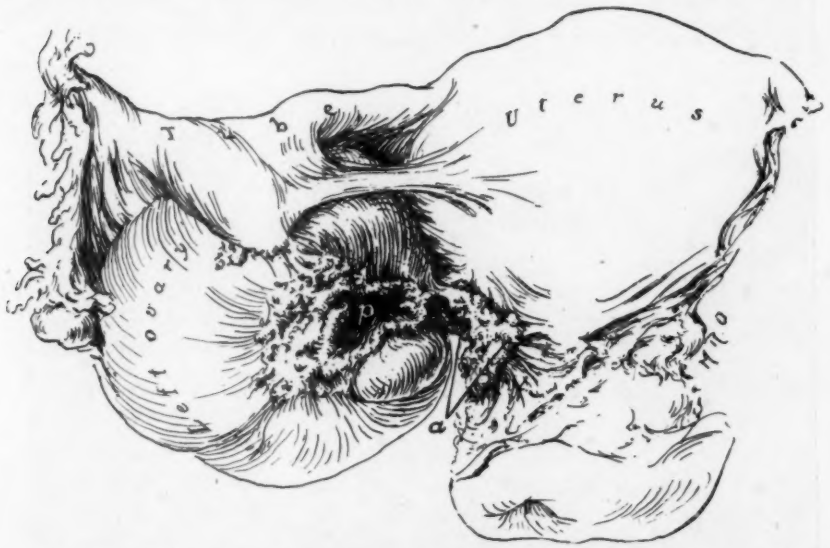

Fig. 28.—Endometrial hematoma (about 4.5 cm. in diameter) of the left ovary, with evidence of a previous perforation and subsequent fusion of the ovary with the posterior surface of the uterus, metaplasia of portions of the epithelial lining of the hematoma, and endometrial invasion of the posterior uterine wall. The patient, unmarried, and aged 35, complained of menstrual pain increasing in severity. The last menstrual flow ceased four days before the operation. Pelvic examination showed the uterus to be retroflexed, with a nodular induration in the cul-de-sac and an adherent mass on the left side. The preoperative diagnosis was an endometrial ovarian hematoma with implantation in the cul-de-sac. The posterior view of the uterus and the ovarian hematoma with the perforation through its inferior wall is shown; × 5/6. The hematoma at the site of perforation (*p*) was fused with the posterior uterine wall, as indicated at (*a*). The histologic structure of the lining of the ovarian hematoma may be seen in Figures 24, 25 and 29, and the invasion of the posterior uterine wall is shown in Figure 30.

implantation theory offers the most rational explanation for the origin of these identical lesions in different situations in the pelvis (Figs. 11 and 12), unless it is proved that menstrual blood and other irritants are capable of stimulating the peritoneal serosa and the surface epithe-

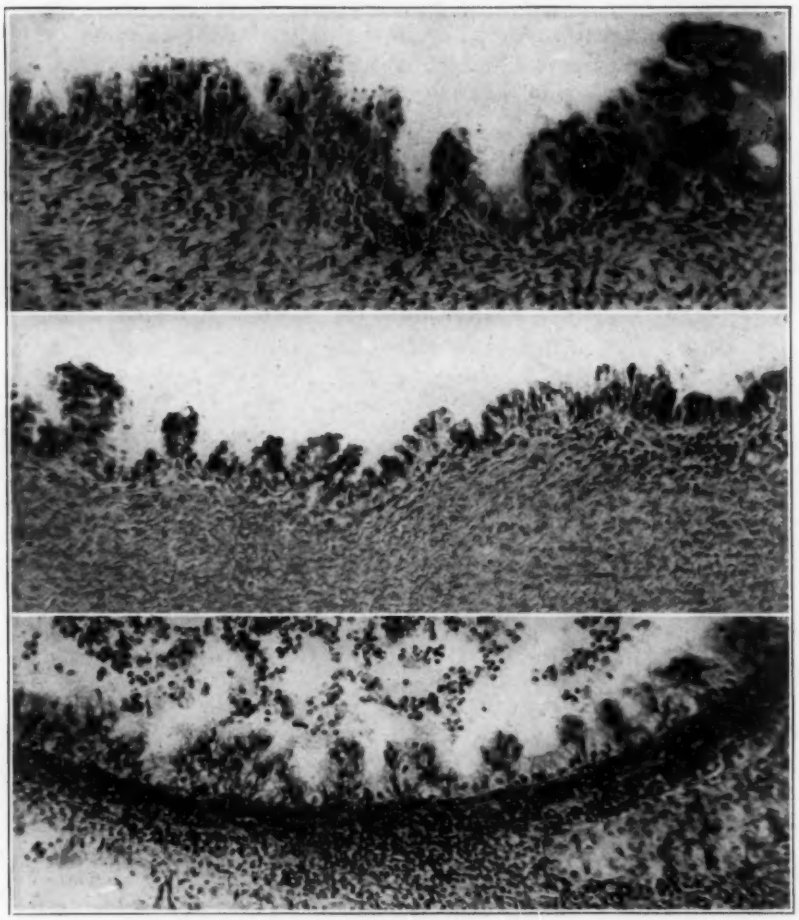

Fig. 29.—Three photomicrographs (× 130), the upper one of a portion of the wall of the benign endometrial ovarian cyst or hematoma (Fig. 28), the second of the malignant portion of the wall of a benign endometrial ovarian cyst with carcinomatous changes (Case 5), and the third of a dilated glandular space in an adenocarcinoma (endometrial) of the body of the uterus. The epithelial lining in the first shows a hyperplasia and also a metaplasia, histologically, less malignant than that shown in the second and more malignant than that shown in the third photomicrograph. There is proof that the conditions presented in the second and third photomicrographs are malignant, but none that those shown in the first one are, except its histologic structure and its similarity to the other two. Does the lesion in the first represent early malignant changes? What would be the life history of such a condition if it had not been removed? If cancer should develop in such case, conditions very similar to those shown in Figures 57 and 58 (Case 5) might be expected.

lium of the ovary to develop into tissue identical with that of the endometrium, both in its histologic structure and function.

In the diagnosis of misplaced endometrial tissue, many factors are of assistance, i. e., its gross appearance, especially as seen at operation (Figs. 13, 14, 16, 17, 18 and 19), its situation (Figs. 11 and 12), associated conditions such as an ovarian hematoma with evidence of a previous perforation (Figs. 28 and 33), or other implants, and, finally, its histologic structure. In consideration of the latter, we look carefully for (1) tissue resembling endometrial stroma about the glands, (2) evidence of hemorrhage (especially indications of previous hemorrhages, as a fresh hemorrhage may be caused by the manipulation of the opera-

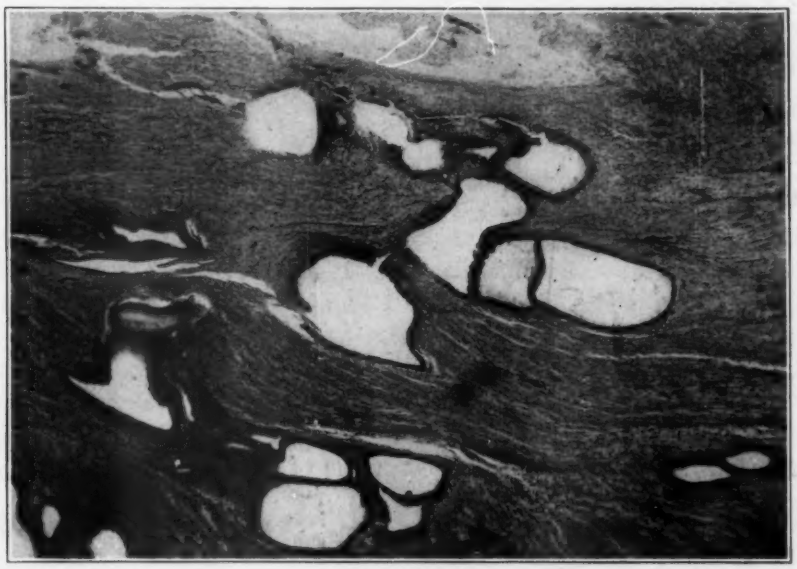

Fig. 30.—The portion of the uterine wall fused with the ovarian hematoma at the site of its perforation, indicated in Fig. 28; × 25. A typical endometrial invasion is present which, after the menopause, might be similar to the benign endometrial invasion of the posterior uterine wall of the uteri associated with ovarian carcinoma in Figures 39, 48, 49 and 53.

tion) and (3) ciliated epithelium. True endometrial glands may occur without any characteristic endometrial stroma about them, without any evidence of a reaction to menstruation and without any demonstrable cilia. I am unwilling to make a diagnosis of endometrial tissue unless some of the glands are accompanied by tissue of the histologic structure of endometrial stroma. While I have often been unable to decide whether a given gland-like structure is of true endometrial or of mesothelial origin, usually after a careful study of the entire specimen

I have been able to decide whether or not an endometrial implantation has occurred. I believe that the histologic diagnosis of misplaced endometrial tissue can be made as definitely as that of many other pathologic lesions, as definitely, for example, as those resulting from tuberculosis. In the latter lesions, one may stain for tubercle bacilli, but, unfortunately, there is no specific stain for any of the elements of endometrial tissue.

It is often impossible to decide whether a given implant is of uterine or of tubal origin. When a gland in a peritoneal or ovarian implant has little or no endometrial stroma about it, contains many ciliated epithelial cells, shows a tendency for the formation of papillae and apparently has not reacted to menstruation, it suggests a tubal rather than a uterine origin.

The extensive endometrial invasion of the various pelvic structures is easily recognized histologically by those familiar with them; this is also true of the changes in the lining of endometrial hematomas of the ovary (Figs. 24, 25 and 26). The latter have been fully described in a previous communication.[5] It may be difficult to diagnose these hematomas in elderly women because the greater portion or all of their epithelial lining may have disappeared, but the pigment in their walls persists (compare Figs. 26 and 55). It is necessary to recognize all of these as foundations for the study of the endometrial origin of ovarian and "peritoneal" carcinomas.

The ninety-eight cases of endometrial implantation and the conditions arising from them, which I encountered this last year, can be classified as:

1. Small implants on both the surface of the ovary or ovaries and on other structures in the pelvis (twenty-eight cases).

2. Peritoneal implants in the pelvis, without any demonstrable ovarian lesion (thirty-four cases).

3. Small implants on the surface of the ovary or ovaries, without any evident peritoneal involvement (eight cases).

4. Ovarian hematomas from 5 mm. to 12 cm. in diameter (twenty-eight cases). The majority of these had apparently perforated and were adherent at the site of perforation to various pelvic structures. They were also associated with other adhesions, apparently resulting from the escape of the contents of the hematoma into the pelvic cavity. Furthermore, there were usually associated with these peritoneal implantations of greater extent and a deeper endometrial invasion of the underlying structures than in the previous groups.

Intestinal implants were present in sixteen patients. The rectosigmoid was involved in nine, the sigmoid in six, the small intestine three times and the appendix once.

This classification furnishes a conception of the benign endometrial lesions which may be present in patients with ovarian carcinoma possibly arising from malignant changes in endometrial tissue in the ovary. It teaches us where to look for these implantations at the time of the operation, and in histologic study of the specimen removed. It also shows the relative frequency of the various types of benign endometrial lesions.

ADENOCARCINOMA OF THE BODY OF THE UTERUS AS A STANDARD FOR COMPARISON IN THE STUDY OF CARCINOMA OF THE OVARY

The histologic study of endometrial carcinoma of the uterus teaches us that it presents a varied structure not only in different specimens but also in different portions of the same specimen. There is also a great variation in the type of the malignant epithelium in different specimens and sometimes in different portions of the same specimen, which is true of all adenocarcinomas.

The very interesting feature of the comparative histologic study of uterine adenocarcinoma and carcinoma of the ovary is the frequent great similarity of the histologic structure of the growths in the two organs, as it is possibly modified by the alterations brought about by the reaction on the part of the ovarian tissue invaded, and its influence both on the type of structure and on the type of cell created by the cancer developing in that tissue. An apparent reaction is the tendency for the development of cysts in the ovary and papillary projections of the cancer into the cavity of these cysts. In many cases, this is due to cancer arising in benign cysts.

REPORT OF CASES DEMONSTRATING THE POSSIBLE ORIGIN OF CANCER IN IMPLANTED ENDOMETRIAL TISSUE, ESPECIALLY IN THE OVARY

Two methods of attacking this problem present themselves: (1) the study of benign endometrial implants and the conditions resulting from them, with the endeavor to find early malignant changes in them, and (2) the study of ovarian carcinoma to ascertain whether or not the carcinoma could have arisen in misplaced endometrial tissue.

The first method is the more natural and more conclusive, but it is also the more difficult as it is analogous to the routine examination of the mucosa of apparently nonmalignant uteri for the purpose of detecting early malignant changes in them. The second method is analogous to the study of obvious cancer of the body of the uterus to ascertain whether or not it arose in the uterine mucosa.

Glandular hypertrophies and changes in the epithelium of apparently benign implants and the conditions arising from them not infrequently occur, and some might be considered "precancerous," or what I prefer

to call potentially malignant, as shown in Figures 29 and 32. A certain number present the same difficulties in regard to an exact diagnosis as the changes sometimes found in the mucosa of the uterine cavity which are not definitely malignant.

While I have carefully studied all my specimens of benign endometrial tissue invading the peritoneum and ovaries, which were not associated with ovarian carcinoma, I have not been able to detect early malignant changes in them other than those similar to the ones shown in Figures 29 and 32, which I am unable definitely to call malignant. I believe, however, that they will be found. On the other hand, the study of the specimens of obvious ovarian carcinoma was much more

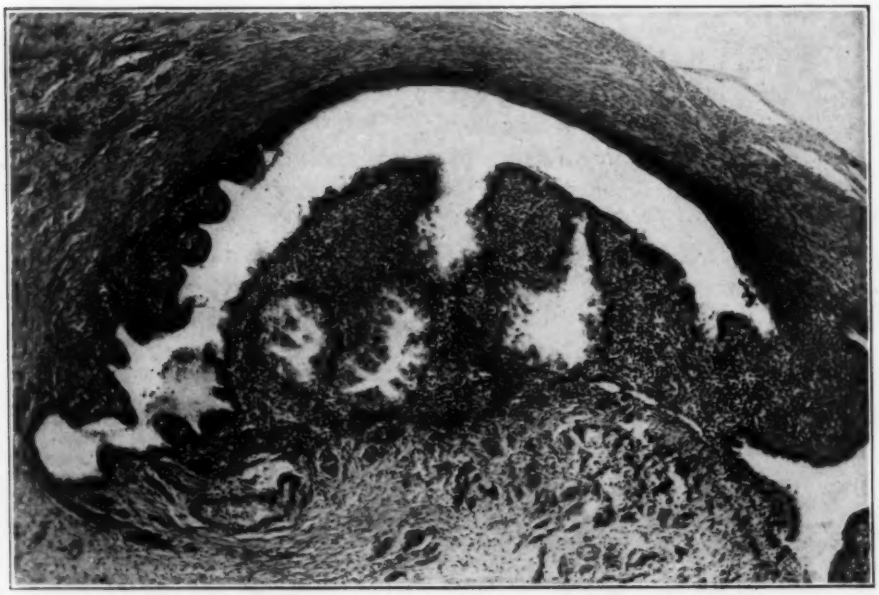

Fig. 31.—A small endometrial cavity of the ovary, showing a typical glandular hyperplasia; ×25. The patient, unmarried, aged 32, complained of dysmenorrhea increasing in severity. The last menstrual flow occurred three weeks before the operation. The uterus was retroflexed with a nodular induration in the cul-de-sac, and the left ovary was enlarged and adherent. The preoperative diagnosis was an endometrial ovarian hematoma with endometrial implantation. At operation, the left ovary was found to be adherent and contained a hematoma, about 2 cm. in diameter, with evidence of a previous perforation on its lateral surface. Implants were present in the cul-de-sac and on the posterior surface of the uterus. The appendix and left tube and ovary were removed, the peritoneal implants excised, the uterus suspended and the cervix dilated. This small endometrial cavity was situated near the lateral surface of the ovary, about 5 mm. from the perforation of the ovarian hematoma. Typical glandular hyperplasia (possibly premenstrual) was present in a portion of its lining. Ciliated epithelial cells were present. Glandular hyperplasia in ectopic endometrial tissue is of quite frequent occurrence.

satisfactory, but, even when cancer was found replacing or even apparently arising from endometrial tissue in the ovary, the question invariably came up as to whether or not it arose in that tissue.

The cases of ovarian carcinoma were studied in the following manner:

1. At the operation, the pelvic contents were carefully examined to ascertain whether or not any benign endometrial implants and the conditions resulting from them were present.

2. If the ovarian tumor was adherent to the posterior surface of the uterus or broad ligament, an attempt was made to remove the uterus, tubes and ovaries without disturbing this relation. The fusion of the ovarian tumor with these structures may have resulted from the carcinoma, from the adhesions resulting from the implantation of endometrial tissue on the surface of the ovary or from the perforation of an endometrial hematoma.

Fig. 32.—A small endometrial cavity of the ovary showing an atypical hyperplasia of a portion of its epithelial lining; × 25, from the same section shown in Figure 31, but nearer the perforation of the large ovarian hematoma. The histologic structure of the epithelial hyperplasia very closely resembles that sometimes found in cancer of the body of the uterus and in malignant ovarian cysts. What is its significance? What would have been its life history in later years had it not been removed? Is it potentially malignant? These are all interesting problems to be solved.

3. Careful drawings were made of the specimen removed. Pieces of tissue were fixed in Zenker's solution and then the entire specimen was placed in formaldehyd solution. When the specimen had hardened, gross serial, cross or sagittal sections were made in order to study the

exact relation of the tumor to the uterus and other structures (if adhesions existed), and also to show the appearance of the tumor in different areas.

4. All tissue suggesting benign endometrial implantation was examined microscopically.

5. Particular attention was paid to the lateral and under surface of the ovaries, whether they were malignant or nonmalignant, as these are the areas most frequently invaded in benign endometrial implantation.

6. Sections from many portions of the tumor were studied, and especial attention was given those showing any variation in their gross appearance.

REPORT OF CASES

CASE 1.—*Endometrial cysts or hematomas of both ovaries, with evidence of perforation of the right one, extensive endometrial implantation of the sigmoid, cul-de-sac and posterior surface of the uterus, "endometrial" papilloma on the surface of the right ovarian hematoma.*

History.—The patient, aged 46, complained of pain in the lower abdomen and severe dysmenorrhea. She had been married for twenty-six years, and had never been pregnant. Menstruation had been regular, moderate in amount and always painful. The amount of pain varied and had been more severe in recent years. The last menstrual flow occurred two weeks before the operation.

Examination.—The pelvic examination showed that the uterus was slightly enlarged, in anteposition and fixed in the pelvis with adherent masses on both sides, the larger on the right, and a nodular induration in the cul-de-sac. The preoperative diagnosis was endometrial ovarian hematoma with implants in the cul-de-sac.

Operation.—The patient was operated on at the Albany Hospital, June 26, 1922. A hematoma of the right overy about 8 cm. in diameter was found, densely adherent to the posterior surface of the right broad ligament. When it was freed, a large amount of chocolate-like fluid escaped. The sigmoid was fused with the posterior surface of the uterus. The left ovary was slightly enlarged and adherent; both tubes appeared patent and normal. A papilloma about 3 cm. in its greatest diameter was attached by a small pedicle to the upper surface of the right ovarian hematoma, lateral to the fimbriated opening of the tube (Fig. 33). The gross appearance of this papilloma was similar to that of those found in certain malignant ovarian cysts. Extensive benign endometrial implants were present on the sigmoid, the posterior surface of the uterus and in the cul-de-sac. The entire uterus, both tubes and ovaries and the appendix were removed. The patient made a satisfactory convalescence and has remained well.

The uterus was slightly enlarged; its mucosa was thickened and a polyp about 1.5 cm. in its greatest diameter was situated in the fundus near the midline. The entire posterior surface of the uterus below the level of the attachment of the ovarian ligaments was invaded by endometrial tissue, in places forming typical so-called "adenomyomas." Both tubes appeared patent and normal. The left ovary was slightly enlarged and contained an endometrial hematoma about 2 cm. in its greatest diameter. The greater portion of the right ovary was replaced by a hematoma about 8 cm. in its greatest diameter, which had previously perforated into the peritoneal cavity through its under and lateral

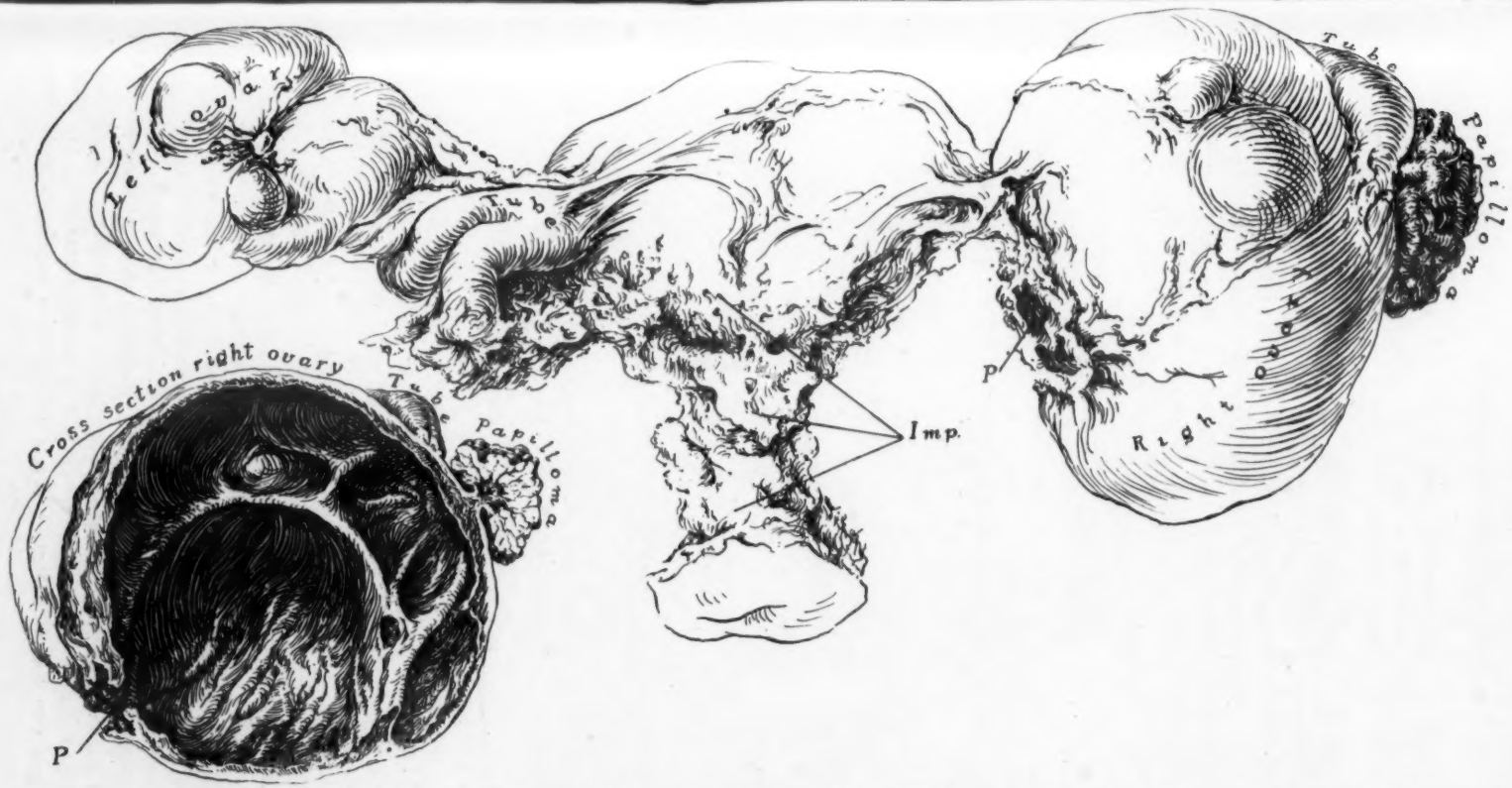

Fig. 33 (Case 1).—Posterior surface of the uterus, tubes and ovaries; × 5/6. The hematoma of the right ovary, 8 cm. in its greatest diameter, was fused with the posterior surface of the right broad ligament, at the site of a previous perforation of the former (indicated by *P*). A cross section of the right ovarian hematoma shows that its cavity is sacculated as though several ovarian hematomas had perforated into each other, or an endometrial hematoma had perforated into follicular cysts. The papilloma was attached by a small pedicle to the superior surface of the ovarian hematoma, just lateral to the fimbriated end of the tube which was adherent to the surface of the hematoma. I was unable to demonstrate that the papilloma arose from fimbriae of the tube attached to the ovary. The possible origin and the structure of the papilloma may be seen in Figures 34 and 35. Both tubes are apparently patent. The left ovary contained a hematoma about 2 cm. in its greatest diameter, which apparently had not perforated. There was an extensive endometrial invasion of the posterior uterine wall similar to that shown in Figure 30 and apparently a similar invasion of the anterior wall of the sigmoid and the tissues of the cul-de-sac. The structure of portions of the wall of the right ovarian hematoma may be seen in Figures 26 and 36.

surfaces. The cavity of the hematoma was sacculated, as though several endometrial hematomas had perforated into each other, or a single endometrial hematoma had perforated into preexisting follicular cysts of the ovary (Fig. 33). The papilloma, about 3 cm. in its greatest diameter, was attached by a small pedicle to the superior surface of the hematoma, lateral to the fimbriated opening of the tube.

Histologically, the endometrial invasion of the posterior uterine wall presented the same endometrial structures found in so-called adenomyomas arising from the invasion of the uterine wall by the mucosa lining its cavity. Ciliated epithelium was not found in the glandular spaces of this invasion.

The histologic structures of the wall of the right ovarian hematoma were typical of the larger endometrial hematomas. In places, the epithelial lining was columnar, with a vascular underlying stroma; in other places, the epithelium was flattened. The greater portion of the lining lacked an epithelial covering (cast off by its reaction to menstruation), and consisted of phagocytic

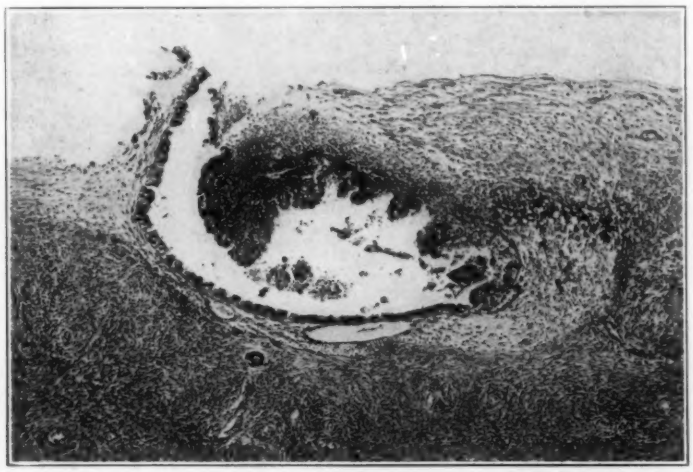

Fig. 34 (Case 1).—An endometrial pocket, in the wall of the right ovarian hematoma, near the base of the papilloma; × 60. The wall of the pocket is lined by cuboidal and columnar epithelium, in places with an underlying stroma similar to that of the endometrium and with hemorrhage into the same. Small papillae are present, projecting into the cavity of the pocket. Ciliated epithelium is also present; one might infer that it arose from the true surface epithelium of the ovary, but the associated conditions would indicate an implantation. The histologic structure of the epithelial lining of the pocket is identical with that covering portions of the papilloma (Fig. 35), and the latter may have arisen from a similar structure.

cells in various stages of preservation, blood pigment and connective tissue (Fig. 26). A few polypoid growths of endometrial tissue were found projecting into the cavity of the hematoma (Fig. 36). Ciliated epithelium was not found in the lining of the hematoma (rarely found in the larger hematomas). Many small endometrial implants were found on the surface of the hematoma (Fig. 34) Ciliated epithelium was found in some of these (more often present in them

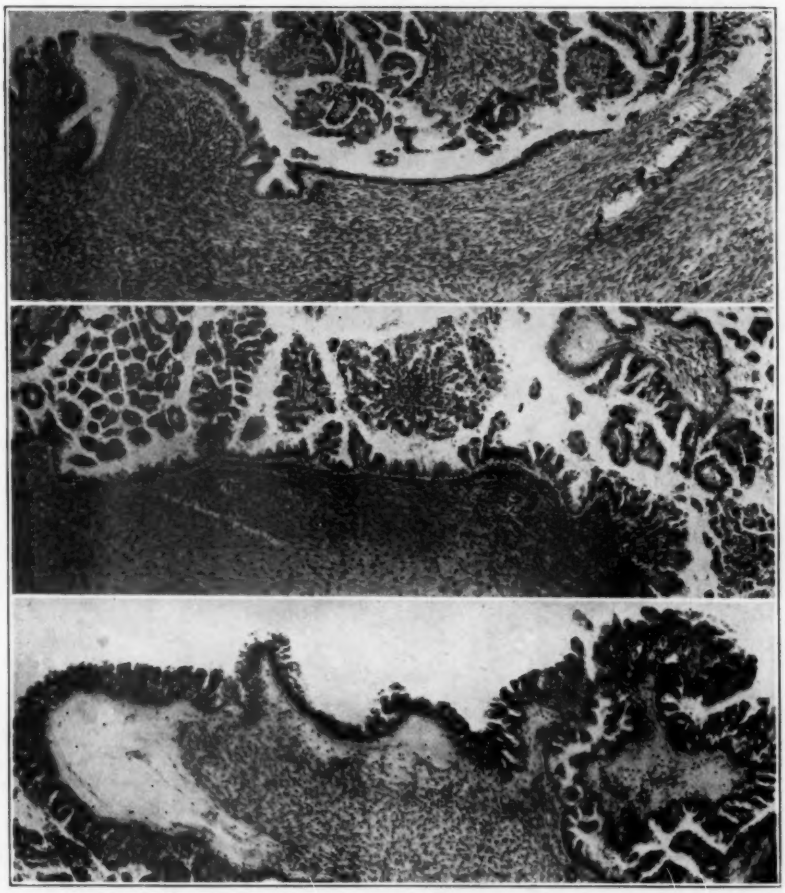

Fig. 35 (Case 1).—Variations in the epithelial covering of the papilloma on the surface of the ovarian hematoma; × 60. The histologic structure of the epithelial covering of a portion of the papilloma shown in the upper photomicrograph is identical with that shown in Figure 34, and also with that found in sections of the fallopian tube. Ciliated epithelium is also present. In the next photomicrograph, this epithelium has undergone a hyperlasia and also a metaplasia with the formation of small papillae. In the third photomicrograph, the hyperplasia and metaplasia are more marked, giving rise to a form of growth often found in ovarian papillomas and carcinomas. This papilloma may have arisen in an implant similar to that shown in Figure 34, although its origin from a tubal fimbria adherent to the ovary or the true surface epithelium of the ovary cannot be excluded.

than in the larger endometrial cysts). In some of these implants, there was a tendency for the formation of papillary growths, suggesting an origin from tubal mucosa (Fig. 34).

Histologically, the papilloma on the surface of the ovarian hematoma suggested an origin either from one of the implants just described, from a fimbria of the tube attached to the surface of the ovary, or from the true surface epithelium of the ovary. In places, normal mucosa with ciliated epithelium, identical in its histologic structure with that of the tube and the smaller implants, was found and one could follow the gradual transformation of this epithelium into the typical semimalignant papilloma (Fig. 35), similar in its histologic structure to those found in certain cysts of the ovary. In a section of the right tube near its fimbriated extremity, a papillary reaction of the epithelium somewhat similar to that found in the papilloma just described was present.

Tissue was not removed from the sigmoid for histologic study, but the gross appearance of the portion adherent to the uterus indicated an endometrial invasion of the intestine similar to that of the posterior uterine wall.

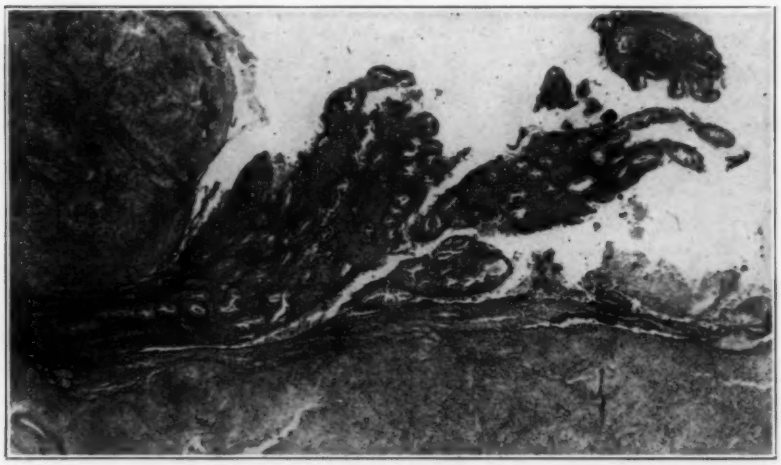

Fig. 36 (Case 1).—Endometrial polyp projecting into the cavity of the large ovarian hematoma; × 25. Histologically, it is a typical uterine polyp. Ciliated epithelium was not found in any of its glands; it may not be present in the glands of a polyp arising from the mucosa of the uterine cavity. Several of these small polyps were found in this specimen, but none of them showed the changes present in the papilloma (Fig. 35) on the surface of the hematoma.

Interpretation of Findings.—My interpretation of the findings in this case is: As a result of the back-flow of menstrual blood into the peritoneal cavity, bits of uterine mucosa and possibly tubal mucosa had been carried into the pelvis, causing primary endometrial implantations on the surface of the peritoneum and the ovaries. Some of the ovarian implants developed into menstruating ovarian cysts or hematomas, one of which perforated, causing secondary implants of endometrial tissue. The papilloma on the surface of the ovarian hematoma arose from "malignant" changes in a primary or secondary implant similar to that shown in Figure 34, from a portion of a fimbria of the tube attached to the ovary, or from the true surface epithelium of the ovary.

My present reaction is that it arose from an implant. Many sections of the wall of the ovarian hematoma about the pedicle of the papilloma were cut, and I was unable to demonstrate its origin from a portion of a fimbria of the tube.

If the patient had not been operated on, peritoneal implantations from the papilloma would probably have occurred.

CASE 2.—*Carcinomatous cyst of the right ovary, of the gross appearance of a multilocular psuedomucinous cystadenoma, showing in a few scattered areas an epithelial lining of the type found in the cervical mucosa. Right hematosalpinx, benign endometrial invasion (senile stage) of the posterior uterine wall and both ovaries, remains of a small endometrial hematoma of the left ovary, the benign endometrial tissue in the right ovary bearing the same histologic relation to the malignant tissue in that ovary as is found in adenocarcinoma of the body of the uterus.*

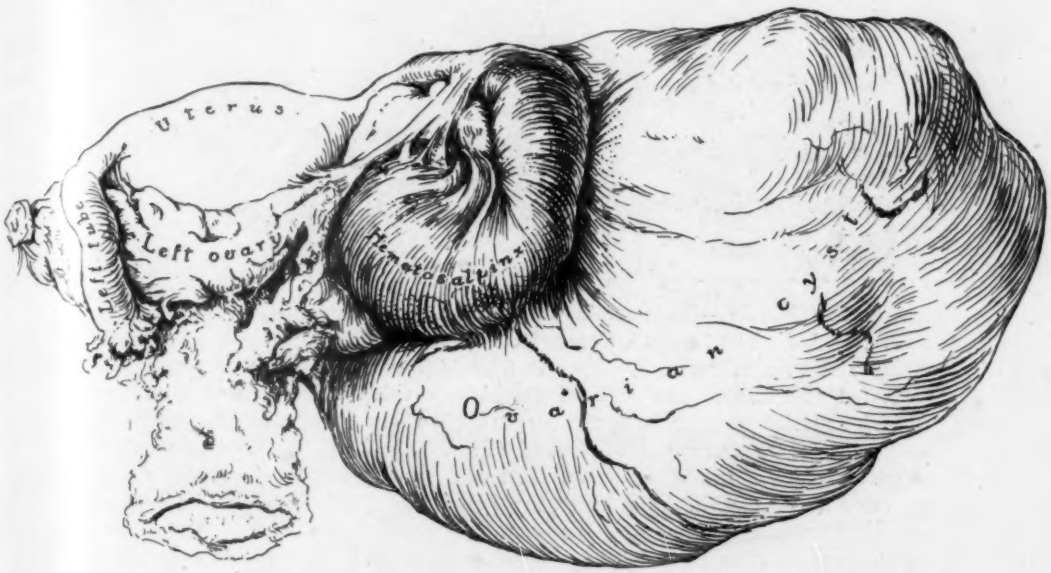

Fig. 37 (Case 2).—Uterus, tubes and ovaries removed at operation without disturbing their relation to each other; × ¾. The right ovarian tumor and hematosalpinx are adherent to the posterior surface of the uterus, as is also the left ovary. The lower portion of the uterus was firmly adherent to the rectosigmoid as the result of benign endometrial tissue implanted on the posterior surface of the uterus, invading that organ and probably also the intestine, a condition that so often occurs in benign endometrial implantations.

History.—The patient, aged 62, complained of uterine bleeding of four weeks' duration. She had had three children, the youngest twenty-seven years before. The menopause occurred at the age of 50.

Examination.—Her general health had been good, and the bleeding was her only complaint. Pelvic examination showed that the uterus was of normal size and position, and fixed in the pelvis, with some induration in the cul-de-sac. A cystic-feeling tumor was present in the right side of the pelvis; this also

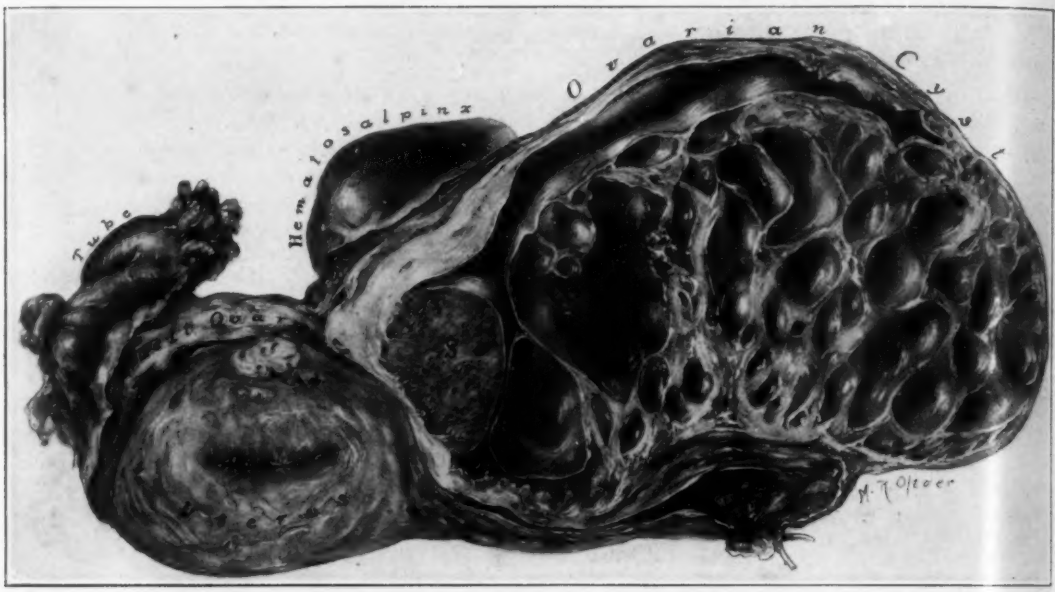

Fig. 38 (Case 2).—Cross section of the uterus and both ovaries, viewed from below; × 5/6. The benign endometrial invasion of the posterior uterine wall, giving rise to a circumscribed endometrial leiomyoma (*Ad*), at this level, is indicated (Fig. 39). The wall of the right ovarian cyst is fused with the posterior uterine wall. A benign endometrial invasion of the portion of the cyst wall adherent to the uterus was found (Figs. 40 and 41). The benign endometrial structure, arising from this invasion, similar to those present in the posterior uterine wall, bore the same histologic relation to the malignant epithelial lining of the cyst that cancer of the body of the uterus bears to the uterine mucosa (compare Figs. 42, 43 and 44). The gross appearance of the cyst is that of a benign multilocular pseudomucinous cystadenoma, and the character of the contents of some of the loculi was similar to that found in these cysts. The greater portion of the epithelial lining of the tumor presented the histologic structure of an adenocarcinoma similar to that found arising in the uterine mucosa (Fig. 45). Scattered throughout, the tumor glands were found partially or wholly lined with epithelium of the cervical type. This was also true of portions of the lining of some of the larger cavities (Fig. 45). The histologic relation between the endometrial invasion of the wall of the cyst and the epithelial lining of the latter (Figs. 40, 41, 42, 43 and 44) is such as to suggest that the cyst might have been of endometrial origin. On the other hand, the carcinoma may have arisen in the epithelial lining of a pseudomucinous cystadenoma (not of endometrial origin) and may have invaded the endometrial tissue in its walls, presenting the appearance of arising in the latter. The latter interpretation is more in accord with our present knowledge of pathologic lesions of the ovary, but is not necessarily the correct one.

Fig. 39 (Case 2).—A section of a portion of the posterior uterine wall and left ovary, showing the endometrial invasion of the uterine wall giving rise to an endometrial leiomyoma with dilated glands (*Ad*) Figure 38; × 10 (compare also with Figs. 30 and 41). The remains of a small endometrial hematoma (*h*) is present in the tip of the left ovary.

Fig. 40 (Case 2).—A portion of the posterior uterine wall and of the ovarian cyst adherent to it; × 25. Benign endometrial tissue is invading both organs.

seemed adherent. The preoperative diagnosis was a malignant ovarian cyst with implantation in the cul-de-sac and a probable carcinoma of the body of the uterus.

Operation.—The operation occurred at the Albany Hospital, June 20, 1923. A cyst of the right ovary was found, which was adherent to the posterior surface of the right broad ligament and the uterus. The right tube was occluded, dilated and also adherent to the posterior surface of the uterus. The left ovary was atrophic and adherent to the posterior surface of the uterus. The left tube appeared patent and normal. The rectum was fused with the posterior wall of the cervix, obliterating the cul-de-sac. There was no free fluid in the peritoneal cavity and no evidence of malignant peritoneal implantations. Both tubes and ovaries and the entire uterus were removed without rupturing the cyst and without disturbing the relations between the ovaries and the uterus (Fig. 37). The appendix was also removed, and the patient made a satisfactory convalescence.

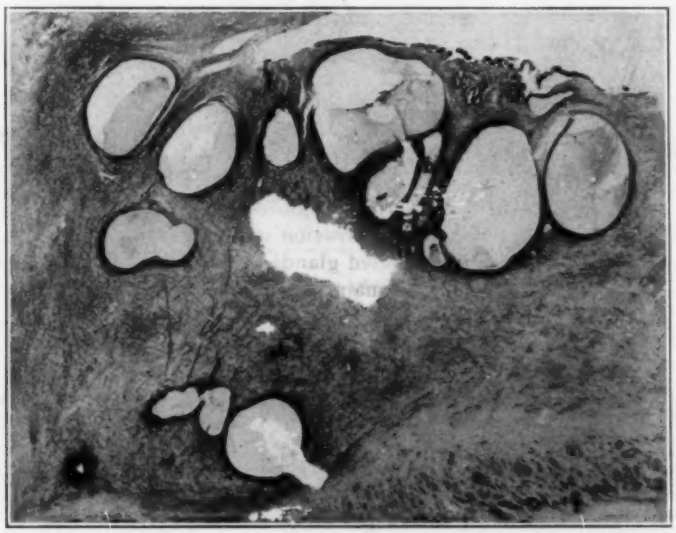

Fig. 41 (Case 2).—A portion of the wall of the ovarian cyst only a few millimeters above the area shown in Figure 40; × 10. A benign endometrial lesion is present with dilated glands histologically identical with those shown in Figure 39 including the presence of ciliated epithelium. Some of these glands were situated just beneath the malignant lining of the ovarian cyst (Fig. 42). A higher magnification of the small gland (*a*) the lumen of which was continuous with the larger gland (*b*) above it, is shown in Figure 43.

The uterus was of normal size. There was benign endometrial invasion (senile stage) of the greater portion of the posterior uterine wall, in places forming circumscribed "adenomyomas" (Fig. 40). A small polyp about 8 mm. in its greatest diameter was present in the fundus of the uterus. The rest of the uterine mucosa was atrophic. The left tube appeared normal. The left ovary was atrophic and contained the remains of a small endometrial hematoma. A hematosalpinx was present on the right side, the walls of which showed a varied histologic structure identical with that of those often found in the larger endometrial ovarian hematomas.

The right ovarian cyst was adherent to the posterior surface of the uterus and the right broad ligament (Fig. 38). Its greater diameter was 12 cm. When the cyst was incised, it presented the gross appearance of a benign multilocular pseudomucinous cystadenoma even to the character of the fluid contents of some of the smaller cavities. Recognizing the extensive endometrial invasion of the uterus and ovaries, I thought that I had an opportunity to determine whether or not this type of cyst could arise from endometrial tissue invading the ovary. I was surprised to find that the greater portion of the cyst was histologically malignant, presenting the picture often found in adenocarcinoma of the body of the uterus. Scattered throughout the tumor and most evident in its distal pole, an occasional gland was found which was partially or wholly lined by benign appearing epithelium of the cervical type. This was also true of small areas in the lining of some of the larger cavities. There was a benign endometrial invasion of the portion of the wall of this cyst adherent to the posterior wall of the uterus (Fig. 40). Benign endometrial structures were present in this portion of its wall similar to those in the posterior uterine wall

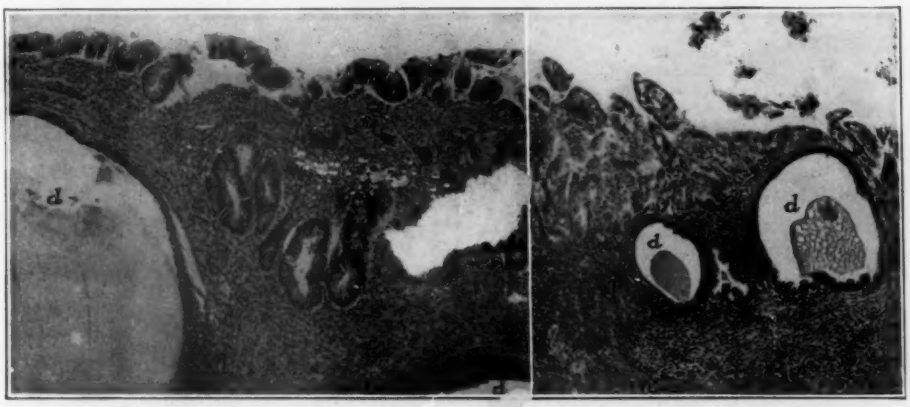

Fig. 42 (Case 2).—Two sections (reduced one sixth from photomicrographs with a magnification of 60 diameters) illustrating the relation between the lining of the malignant ovarian cyst and the dilated endometrial glands (left) shown in Figure 41, as compared with that found in a section of adenocarcinoma of the body of the uterus (right), in which the carcinoma is spreading over the surface of the endometrium. In the latter, dilated glands (*d*) are also present in the uterine mucosa. The histologic relation between the malignant growth and the benign endometrial tissue in the two sections is similar, and suggests a similar origin for the two carcinomas. On the other hand, the endometrial tissue in the ovary may have been invaded by carcinoma from another source (legend of Fig. 38).

(compare Figs. 40 and 41). These structures bore to the malignant lining of the cyst the same histologic relation that adenocarcinoma of the body of the uterus does when the malignant growth comes in contact with the benign uterine mucosa (Fig. 42). The carcinoma was either invading and replacing this benign endometrial tissue or was arising from it, as seen in adenocarcinoma of the body of the uterus. In other portions of the ovary, it was apparently arising from epithelium similar to cervical epithelium and that lining pseudomucinous cystadenomas.

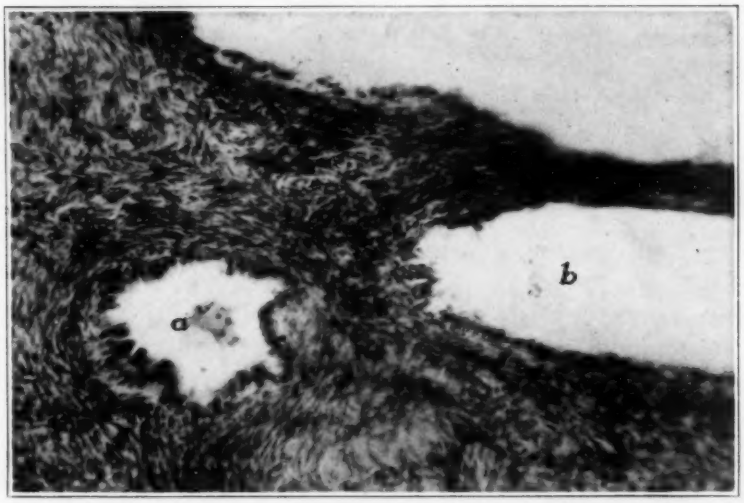

Fig. 43 (Case 2).—The small gland (*a*) shown in Figure 41; × 130. The lumen of this gland is continuous with that of the larger gland (*b*) above it (to the right of illustration). The latter is apparently a dilated benign endometrial gland. The former (*a*) shows a hyperplasia, and also a metaplasia of a portion of its epithelial lining suggesting malignancy.

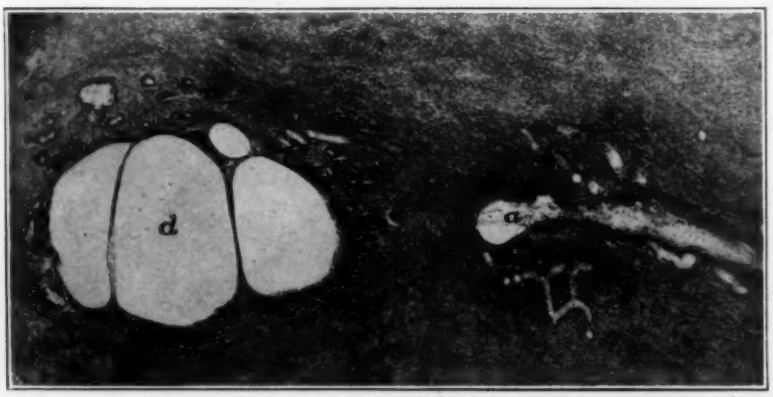

Fig. 44 (Case 2).—Histologically benign endometrial glands surrounded and invaded by cancer; × 25. This section is from a portion of the ovary only a few millimeters from that shown in Figures 42 and 43. The lumen of the glands (*a* and *d*) was shown to be continuous. The epithelial lining of *a* was histologically benign. The dilated portion (*d*) of the gland is surrounded by adenocarcinoma, and in places the cancer is replacing or arising from the epithelium of the gland.

Interpretation of Findings.—The interpretations of all of the findings in this specimen are difficult. Several years ago, endometrial implantations had occurred on the posterior surface of the uterus, in the cul-de-sac and on the surface of both ovaries (Figs. 38, 39 and 40). A small endometrial hematoma had developed in the left ovary, the remains of which were still present. The endometrial tissue developing on the surface of the right ovary caused it to become fused with the posterior surface of the uterus and the right broad ligament. It had invaded the ovary (Fig. 40) and may have given rise to some type of ovarian cyst, as suggested by the intimate relations between the benign endometrial tissue in the ovary and the lining of the malignant cyst (Fig. 41). The gross appearance of the growth, as well as the histologic structure of portions of the malignant cyst suggest that this cyst primarily may have been a pseudomucinous cystadenoma. Could it have arisen from endometrial tissue in

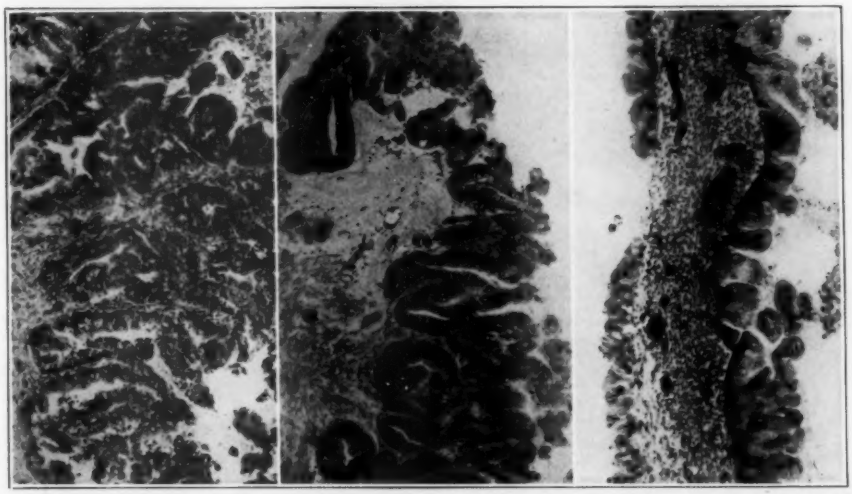

Fig. 45 (Case 2).—Different portions of the malignant ovarian cyst; × 60. The section at the left shows a type of adenocarcinoma often found in cancer of the uterine mucosa. The one at the right shows epithelium of the cervical type such as is present in pseudomucinous cystadenomas, and it suggests that the carcinoma may have arisen in the lining of such a cyst. The photomicrograph in the center presents a histologic structure intermediate between the other two. The difficulties of arriving at a correct interpretation as to the relation between the benign endometrial tissue and the cancer in the tumor are presented in previous illustrations, and in the report of Case 2 in the text.

the ovary? I have never been able to demonstrate the origin of this type of cyst from endometrial tissue. The close resemblance of the lining of these cysts to that of the cervical mucosa suggests this possibility.

Three possibilities as to the origin of the carcinoma in this specimen and its relation to the epithelial structures in the ovary present themselves.

1. The carcinoma arose in the epithelial lining of a small pseudomucinous cystadenoma, which may or may not have been of endometrial

origin, and had invaded the benign endometrial tissue of the ovary, presenting the appearance of arising from it. This is apparently the most acceptable explanation, with our present knowledge of pathologic lesions in the ovary.

2. The carcinoma arose in both the epithelial lining of the cystadenoma and in the endometrial tissue.

3. The cyst arose from endometrial tissue and presented the histologic structure of both a malignant tumor and a cystadenoma.

Fig. 46 (Case 3).—Uterus, tubes and right ovary and a portion of the left ovarian cyst, indicating their relation to each other prior to the operation; × 5/6. The ovarian cyst was adherent to the lower portion of the posterior layer of the left broad ligament and as indicated (*t*), was torn when it was freed. The right ovary is adherent to the posterior surface of the uterus, as was the left ovary in Figure 37. The lower portion of the posterior surface of the uterus was fused with the rectosigmoid. The former, and also probably the latter were invaded by benign endometrial tissue. A circumscribed endometrial leiomyoma arising from this invasion is indicated at *Ad* (see also Fig. 48).

Malignant peritoneal implantations had not occurred because the cancer had developed in epithelial tissue within the ovary, giving rise to a malignant cyst, and the latter had not ruptured nor had the cancer

extended through its wall. The uterine bleeding probably arose from the polyp. The nodular induration in the cul-de-sac was apparently due to benign endometrial implantations diagnosed as malignant prior to the operation.

CASE 3.—*Large carcinomatous cyst of the left ovary, benign endometrial invasion (senile stage) of the posterior uterine wall and both ovaries. A portion of the cyst was lined by epithelium histologically similar to that lining some*

Fig. 47 (Case 3).—View of a portion of the inside of the ovarian cyst (× 5/6), showing the malignant growths projecting into the cavity of the cyst, and also the tear in the wall of the cyst caused by freeing it from the posterior layer of the left broad ligament (Fig. 46).

benign endometrial cysts and also serous cystadenomas. The papillary carcinoma arose in this epithelium.

History.—The patient, unmarried, and aged 61, complained of abdominal distention. The menopause had occurred at the age of 53. She had noticed

Fig. 48 (Case 3).—The circumscribed endometrial leiomyoma indicated by *Ad* in Figure 46; × 10. This, I believe, arose from the invasion of the uterine wall from endometrial tissue implanted on the surface of the uterus and the subsequent reaction of the myometrium (compare Figs. 4 and 40).

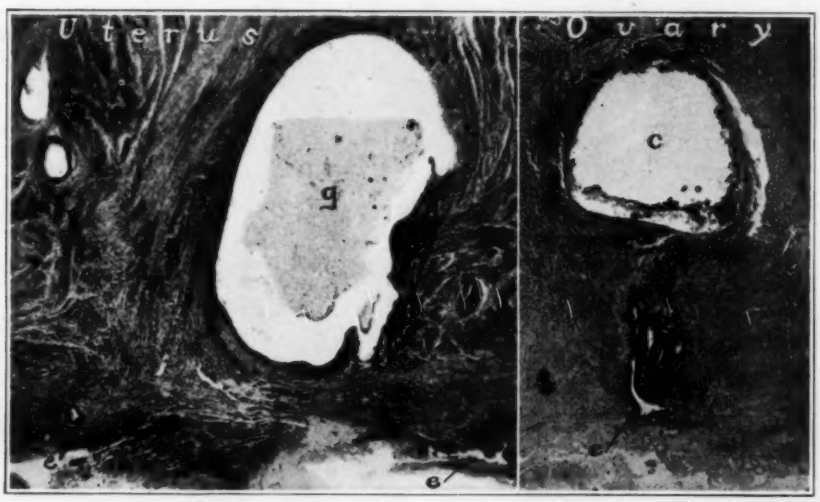

Fig. 49 (Case 3).—Endometrial invasion (senile stage) of the posterior uterine wall and also of the lateral surface of the right ovary, giving rise to dilated glands or small cysts in the wall of the uterus and in the ovary; × 25. A similar glandular dilatation is often present in the mucosa lining the cavity of the senile uterus. Endometrial tissue can be seen on the surface of the uterus (*e*, *e*) and the dilated gland (*g*) apparently arose from this invasion. A similar invasion of the ovarian tissue is indicated by the pointer (*e*), and the small cyst (*c*) apparently arose from this invasion. An occasional ciliated cell was found in the epithelial lining of the cyst (*c*), but none in the dilated gland or cyst (*g*). Ciliated cells were not found in the sections of the senile uterine mucosa, and only a few in the sections of the tubal mucosa. Cilia were not found in all of the dilated glands shown in Figures 1 and 2. The lesions in the two organs are apparently the same, and it could be inferred that they had a similar origin.

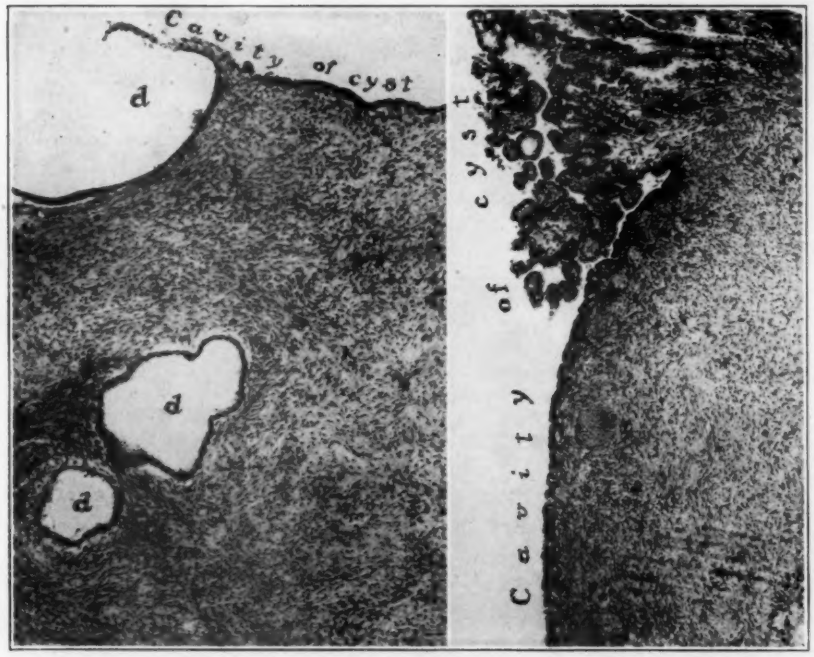

Fig. 50 (Case 3).—Two photomicrographs (×60), indicating the possible origin of the cyst (Fig. 46) from endometrium-like glands in the ovarian tissue, and also the development of a papillary tumor from the lining of the cyst. Endometrium-like glands, similar to those shown in Figure 49, were found in the portion of the wall of the cyst which was adherent to the broad ligament. This is the portion of the ovary which we would expect to have been invaded by implanted endometrial or tubal tissue. These glands had apparently invaded the ovarian tissue from its surface. They were not accompanied by endometrial stroma, but a few ciliated cells were found in their lining. Histologically, they were similar to some of the dilated glands shown in Figure 1 of the mucosal invasion of the uterine wall and also with some of those arising from implanted endometrial tissue in Figures 2 and 4. Their origin from the true surface epithelium of the ovary cannot be excluded, but in view of the associated conditions of this specimen (Figs. 48 and 49) it would seem that they were probably of implantation origin (see text). In the photomicrograph at the left one of the dilated glands (torn) lies just beneath the epithelial lining of the cyst. The latter, in this section, is histologically benign, similar to he epithelial lining of the glands, even to the presence of an occasional ciliated cell. At the right is shown a papillary outgrowth arising in the cyst wall. The epithelial lining of the cyst, which in this section appears benign, is continuous with the epithelial covering of the papillae.

that her lower abdomen had seemed full for about nine months. This fullness had increased of late, and there had been some frequency of urination.

Examination.—A cystic tumor filled the lower abdomen to the level of the umbilicus. The pelvic examination showed the uterus to be anterior to the tumor and fixed in the pelvis, with induration in the cul-de-sac. The preoperative diagnosis was a malignant ovarian cyst with implantation in the cul-de-sac.

Operation.—The operation occurred at the Albany Hospital, July 27, 1923. The cyst (of the left ovary) was adherent to the lower posterior surface of the left broad ligament and was torn in attempting to free it (it should have been aspirated first). The right ovary was atrophic and adherent to the posterior surface of the uterus. The rectosigmoid was fused to the posterior surface of the cervix as in endometrial implantation. There was not any gross evidence

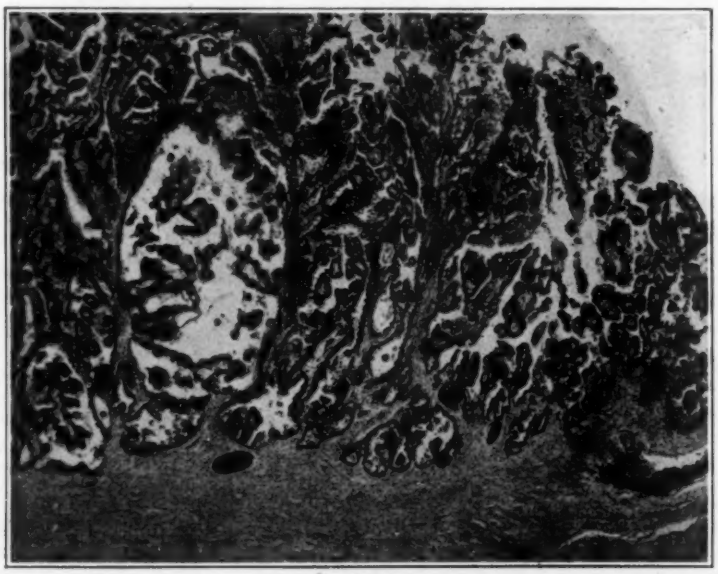

Fig. 51 (Case 3).—A portion of one of the papillary masses projecting into the cavity of the cyst; × 60. Histologically, it is papillary adenocarcinoma, similar to that sometimes arising in the uterine mucosa. The benign endometrial invasion of the posterior wall of the uterus and both ovaries (Figs. 48, 49 and 50), the apparent origin of the ovarian cyst from tubules or glands resulting from this invasion (Fig. 50) and the development of the papillary adenocarcinoma from the epithelial lining of the cyst suggest that the ovarian carcinoma is probably an endometrial carcinoma developing in an endometrial cyst of the type of a serous papillary cystadenoma, rather than in a serous papillary cystadenoma which had arisen from the true surface epithelium of the ovary (see text).

of malignant peritoneal implantations. Both tubes and ovaries, the entire uterus and appendix were removed (Fig. 46). The patient made a satisfactory convalescence.

The uterus was normal in size. There was benign endometrial invasion of the greater portion of the posterior uterine wall, in places forming circum-

scribed "adenomyomas" (Figs. 46 and 48) as in Case 2. Both tubes appeared normal. The right ovary was atprohic and histologically showed benign endometrial invasion (possibly of tubal origin) from its lateral surface (Fig. 49). The wall of the left ovarian cyst was thin, and papillary masses of various sizes projected into its cavity (Fig. 47). These were most numerous in the dependent portion of the cyst (patient standing). Gland-like spaces of endometrial (possibly tubal) type were found in the portion of the wall of the cyst which was adherent to the left broad ligament. A few ciliated cells were found in some of the dilated glands. The epithelial lining in portions of the cyst wall was histologically similar to that lining portions of some benign endometrial cysts and also similar to that of serous cystadenomas. Even a few ciliated cells were found. The papillary growths arose from this epithelium (Fig. 50). In places, typical adenocarcinoma was present (Fig. 51) and, in a few areas, carcinoma of the solid type.

Ciliated epithelium was not found in the glands of endometrial tissue in the posterior uterine wall or in the atrophic mucosa lining the uterine cavity and in only a very few in the tubal mucosa. In places there was an invasion of the wall of the uterus by the mucosa lining its cavity, in one place to a depth of 8 mm., forming a so-called adenomyoma.

Interpretation of Findings.—My interpretation of the findings in this case is as follows: Several years ago, as a result of a back-flow of menstrual blood into the peritoneal cavity, bits of uterine and possibly tubal mucosa were carried into the pelvis, causing implantations on the posterior surface of the uterus and on the ovaries. An endometrial cyst of the left ovary developed from the endometrial or tubal invasion (the epithelial lining of the cyst resembled that of the tube more than that of the uterus) of that organ, and possibly perforated, the cyst becoming adherent to the posterior surface of the left broad ligament. Secondary implantations may have resulted at that time. All these endometrial structures became atrophic at the time of menopause. Recently, a papillary-carcinoma developed in some portion of the epithelial lining of the remains of the endometrial cyst. The induration in the cul-de-sac was due to benign endometrial implantations. Were it not for the evidence of endometrial implantations invading the posterior surface of the uterus and the opposite ovary (Fig. 49), causing glandular elements of the same histologic structure as those giving rise to the ovarian cyst (Fig. 50), one would infer that the cyst was primarily a serous papillary cystadenoma, which had arisen from the true surface epithelium of the ovary.

Case 4.—*Large carcinomatous cyst of the left ovary, benign endometrial invasion (senile stage) of the posterior uterine wall and the left ovary, the cavity of the cyst communicating with a pocket in its wall, which was lined by benign endometrial tissue in portions of which cancer was replacing or arising from the histologically benign endometrial epithelium. There was evidence of a recent perforation of the malignant cyst and malignant implants were present on the peritoneum and in the omentum.*

History.—The patient, aged 56, complained of abdominal distention and a loss of thirty pounds (13.6 kg.) in weight in four months' time. She had been married, but had never been pregnant. The menopause occurred at the age of 50. A cystic tumor filled the lower abdomen, extending just above the umbilicus. The tumor seemed adherent, and there was marked induration in the cul-de-sac. The preoperative diagnosis was a malignant ovarian cyst with implantation in the cul-de-sac. The operation occurred at the Albany Hospital

Sept. 16, 1923. When the abdominal cavity was opened, the peritoneum was found to be stained a dirty grey color (evidence of a perforation of the cyst) Nodules of implantation carcinoma were found on the peritoneum and in the omentum. The cyst was densely adherent to the posterior surface of the uterus and to the left broad ligament. The cyst was first aspirated. Both tubes and ovaries and the entire uterus were removed without disturbing the attachment of the cyst to the posterior surface of the uterus and the left broad ligament. She had a stormy postoperative convalescence, but finally recovered from the operation.

The uterus was normal in size. There was a benign endometrial invasion of the posterior uterine wall, fusing it to the wall of the left ovarian cyst, such

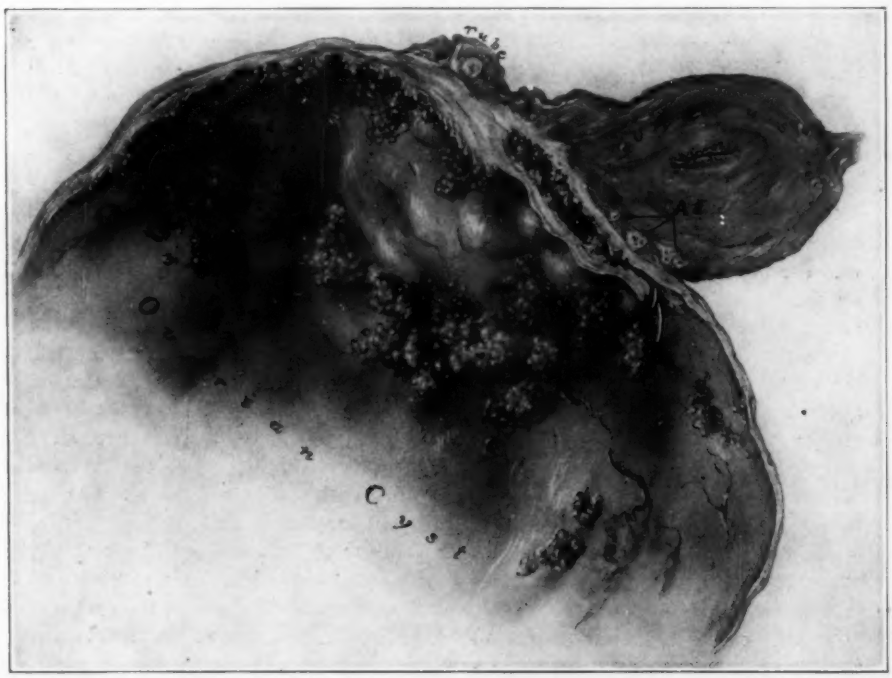

Fig. 52 (Case 4).—Cross section of the uterus, left broad ligament and the portion of the carcinomatous cyst fused with them; × 5/6. Small circumscribed endometrial leiomyomas (*Ad*) arising from the invasion of the posterior uterine wall are shown, as well as the small partially collapsed cyst (indicated by an arrow) communicating with the cavity of the larger one. The smaller cyst is represented semidiagrammatically.

as is often found in benign endometrial ovarian hematomas. The right ovary was atrophic, and neither benign endometrial nor malignant implantations were found on it. Both tubes were apparently patent and bits of cancer were found lying free in the lumen of the right one. Malignant implants were present on the peritoneal surface of the right tube, on the posterior surface of the right broad ligament and in the cul-de-sac. The wall of the left ovarian cyst was thin, and papillary masses of various sizes projected into its cavity. These were

more numerous in the dependent portions of the cyst (patient standing). Sections, from any portions of the wall of the large cyst, were examined in order to determine if any benign endometrial tissue was present. A histologically benign epithelial lining was found in only two sections and in only a very small area of each section (Fig. 55). From these areas alone, I would hesitate to make a diagnosis of endometrial tissue, although a similar lining is often present in portions of known endometrial cysts and was also present in portions of the smaller cyst that communicated with this one, and which was evidently an endometrial cyst (Figs. 53 and 54). In places where an epithelial lining was lacking, the wall of the cyst contained pigment (blood), both intracellular and in its tissues, thus presenting a picture identical with that often found in endometrial cysts (compare Fig. 55 with Fig. 26). For these reasons, I believe

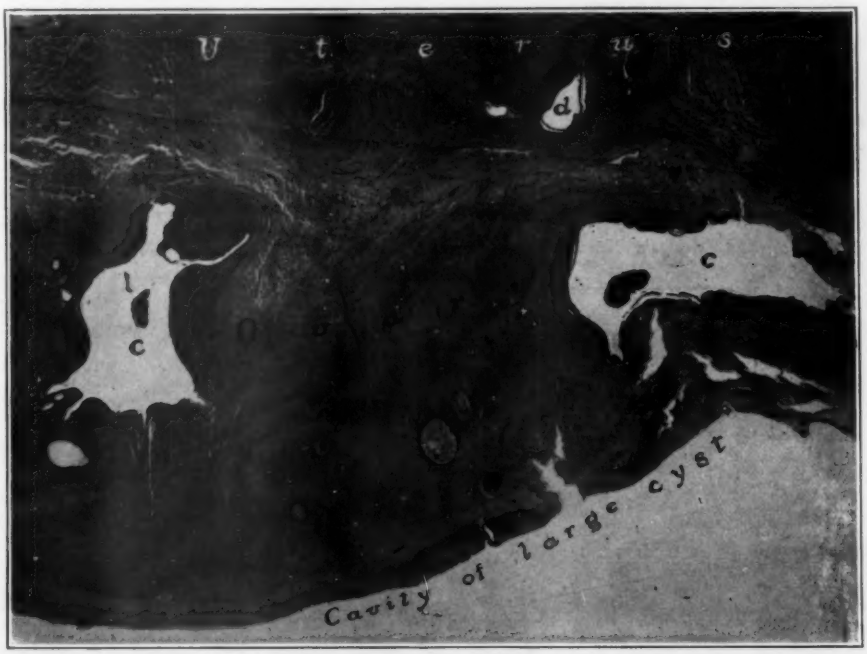

Fig. 53 (Case 4).—A portion of the cyst wall that is fused with the posterior wall of the uterus (Fig. 52); × 10. The glandular structures arising from the endometrial invasion of the uterine wall are shown in the upper third of the illustration; one of these is dilated (*d*). Similar structures are present in the walls of the cyst (the lower two-thirds of the illustration); the cavities (*c, c*) represent sections of portions of the partially collapsed smaller cyst (Fig. 52), which communicate with each other and with the larger cyst. In this situation, the lining of these cavities is histologically identical with that of the dilated gland in the uterine wall and with that of the senile mucosa lining the uterine cavity (Fig. 54). Ciliated epithelium was not found in any endometrial tissue of the specimen including the uterine mucosa. The portion of the larger cyst shown in this section lacks an epithelial lining, the pigmented inner layer of the wall having the same histological structure (Fig. 55) as that of the benign endometrial cyst shown in Figure 26.

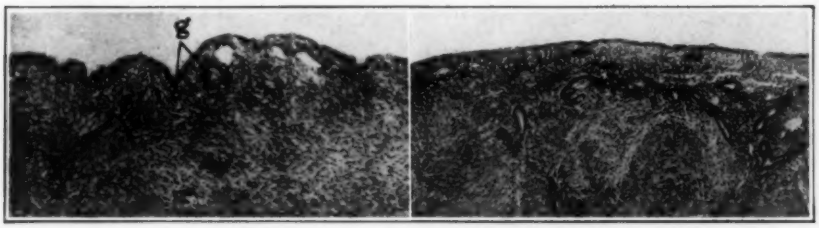

Fig. 54 (Case 4).—A portion of the wall of the small collapsed endometrial cyst of the ovary (left), shown in Figures 52 and 53, and the other (right) of the mucosa lining the uterine cavity (same case); × 60. The cavity of this portion of the cyst is lined with columnar epithelium, with underlying tissue resembling endometrial stroma and containing small glands (*g*). The histologic structure of this portion of the cyst wall is identical with that of the senile and atrophic uterine mucosa shown at the right. The histologic relation between the benign and malignant epithelial lining of this cyst is indicated in Figure 27.

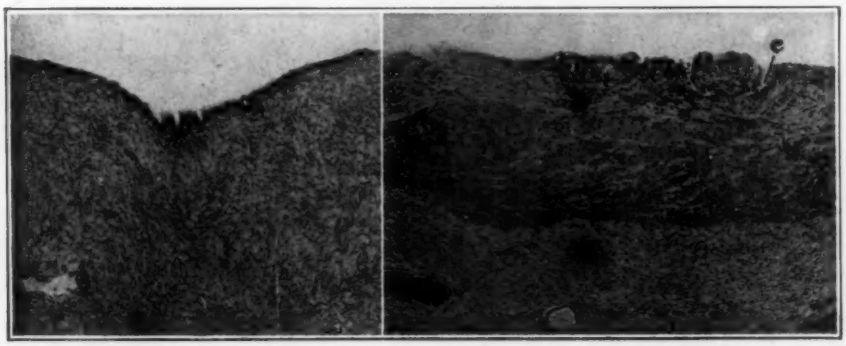

Fig. 55 (Case 4).—Different portions of the wall of the larger malignant cyst; × 60. The one at the left is lined with histologically benign cuboidal and columnar epithelium without any definite endometrial stroma beneath it, but, histologically, it is identical with that sometimes present in portions of known endometrial cysts and also identical with that of portions of the smaller ovarian cyst shown in Figure 53. At the right is shown a higher magnification of a portion of the section of the wall of the larger cyst shown in Figure 53. The epithelial lining has been cast off, except for that present in a slight indentation (*e*). This portion of the wall of the cyst contains pigment (blood), intracellular and free in the tissues of the cyst wall, and presents a histologic picture identical with that often found in portions of benign endometrial cysts (compare Fig. 26). For the foregoing reasons and because of the fact that the cavity of the larger cyst communicated with that of the smaller one (the latter easily recognized as endometrial), I believe that the larger one was primarily a benign endometrial cyst.

that the larger cyst was originally a benign endometrial cyst similar to that of the smaller one with which it communicated. The carcinoma arising in the lining of the cyst was an adenocarcinoma (Fig. 56), in places forming papillary growths.

There was a benign endometrial invasion of the cyst wall similar to that of the posterior uterine wall in the portion of the cyst which was fused with the uterus. An irregular pocket or smaller cyst, about 4 cm. in its greatest diameter, was situated in this portion of the cyst wall, and communicated with the cavity of the larger cyst (Fig. 52). The deeper portion of the smaller cyst was lined by tissue histologically identical with that lining the uterine cavity and the benign endometrial invasion of the posterior uterine wall from its peritoneal surface (Figs. 53 and 54). In the portions of this endometrial cyst, malignant epithelium was found replacing or arising from the benign endometrial lining (Fig. 27), a condition occurring in endometrial carcinoma of the body of the uterus.

Ciliated epithelium was not found in any of the misplaced endometrial tissue, nor in two sections of the mucosa of the uterine cavity, but was present in the tubal mucosa.

Fig. 56 (Case 4).—A portion of the wall of the smaller cyst, showing typical papillary adenocarcinoma identical with that present in portions of the wall of the larger cyst and also identical with that sometimes found in adenocarcinoma of the body of the uterus; × 60. The benign endometrial invasion of the posterior wall of the uterus and left ovarian cyst, which was fused with the uterus and left broad ligament, a condition occurring in endometrial implantation, the histologic relation between the epithelial lining of the benign endometrial tissue in the ovary and the malignant epithelial lining of the cyst (Fig. 27) and the type of growth arising from the latter indicate that the ovarian carcinoma is an endometrial carcinoma, arising in the epithelial lining of a benign endometrial cyst of implantation origin.

Interpretation of Findings.—My interpretation of the findings in this case is as follows: Sometime during the menstrual life of the patient menstrual blood escaped into the peritoneal cavity through the tubes, carrying with it bits of uterine mucosa. The latter caused endometrial implants on the posterior surface of the uterus and on the surface of the left ovary. The endometrial invasion of the ovary was followed by the development of an endometrial cyst. The latter probably perforated, causing secondary implantations and the fusion of the cyst wall with the posterior surface of the uterus and the left broad

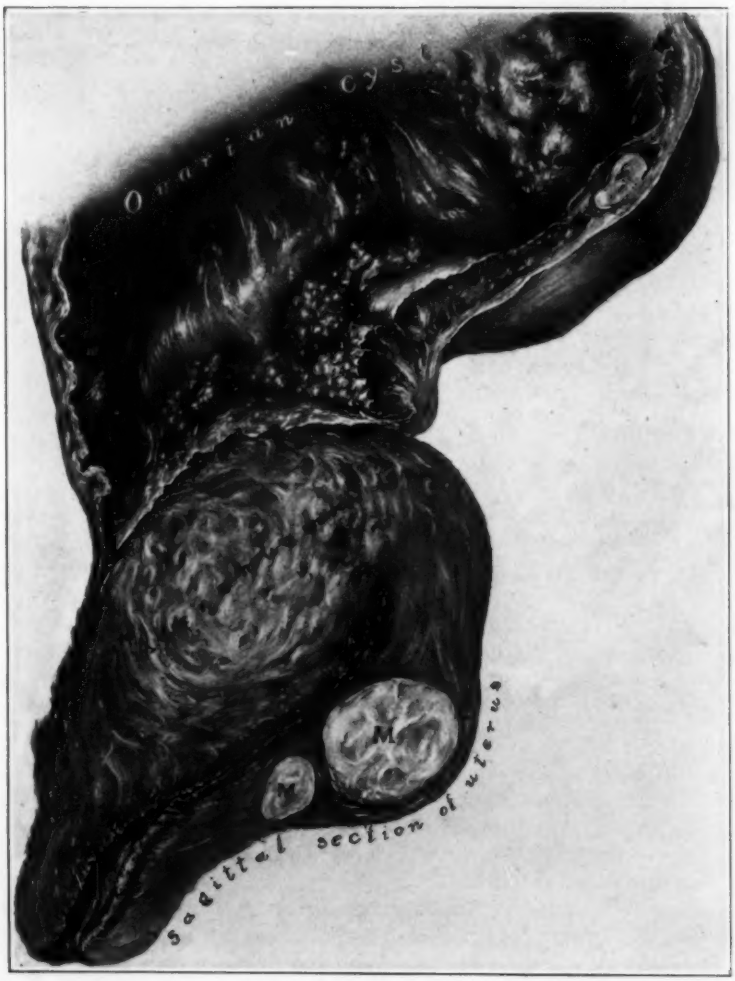

Fig. 57 (Case 5).—Sagittal section of the uterus and portion of the ovarian cyst adherent to it; × 5/6. The wall of the ovarian cyst varies in thickness, being thinnest where it is fused with the posterior uterine wall. A papillary adenocarcinoma of endometrial type has arisen from its lining and is shown projecting into the cavity of the cyst and invading its walls. The "adenomyoma" apparently arose from endometrial tissue invading the posterior uterine wall through perforations of the endometrial cyst (Figs. 59 and 60). Two small leiomyomas (*m* and *m*) are shown in the anterior wall of the uterus.

ligament, such as so often occurs in benign endometrial cysts. All these endometrial structures at the menopause underwent the same involuntary changes as the uterine mucosa. Recently, a malignant ovarian cyst developed from these benign endometrial structures in the left ovary. This cyst had perforated, causing malignant implantations in the peritoneal cavity. The induration in the cul-de-sac was caused chiefly by these malignant implantations.

CASE 5.—*Large endometrial cyst of the left ovary with evidence of perforation and fusion of the cyst wall, at the site of perforation, with the posterior uterine wall; implantation endometrial invasion of the posterior wall of the uterus giving rise to an extensive "adenomyoma," which was not connected with the mucosa lining the uterine cavity; adenocarcinoma of endometrial type arising in the endometrial lining of the cyst.*

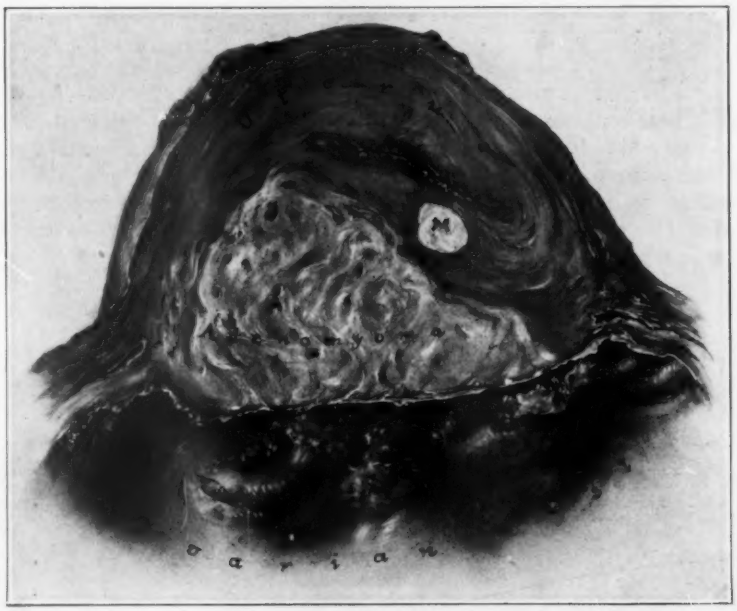

Fig. 58 (Case 5).—Cross section of the uterus, and a portion of the wall of the endometrial ovarian cyst with carcinoma arising in its endometrial lining; × 5/6. This portion of the cyst wall is fused with the posterior wall of the uterus, probably as the result of previous perforations. The "adenomyoma" apparently arose from endometrial tissue escaping through the perforations and invading the uterus (Figs. 59 and 60), and the carcinoma, from malignant changes in its endometrial lining.

History.—The patient, unmarried and aged 46 (not yet having reached the menopause), complained of abdominal pain and distention. She had had three attacks of severe abdominal pain in May, June and July, and was admitted to the Albany Hospital Oct. 22, 1923. The lower abdomen was distended by a cystic-feeling tumor extending above the level of the umbilicus. The pre-operative diagnosis was an ovarian cyst. The patient was operated on by Dr. Arthur W. Elting. I witnessed the operation and recognized the condition

Fig. 59 (Case 5).—A portion of the wall of the ovarian cyst which is fused with the posterior uterine wall (Figs. 57 and 58); reduced a little more than one-fifth from a photomicrograph with a magnification of 8 diameters. The cyst wall is thin and is lined in part by typical uterine mucosa, especially at the right. At *a* this mucosa has extended through the wall of the cyst and has invaded the underlying uterine wall giving rise to a so-called adenomyoma. At *b*, typical uterine mucosa has invaded the uterus beneath the wall of the cyst. The portion of the wall of the cyst between *a* and *b* has apparently been stripped off during the operation.

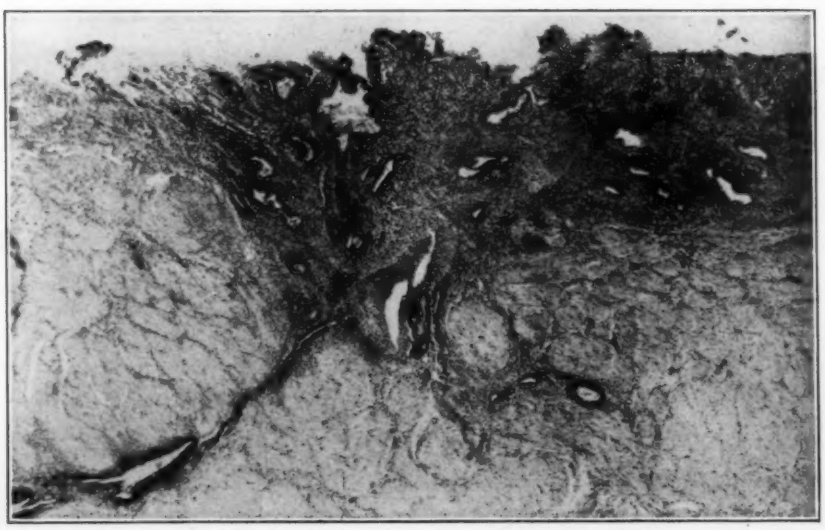

Fig. 60 (Case 5).—Another portion of the posterior uterine wall at an apparent site of perforation of the ovarian cyst, which had become fused with the uterus; ×25. The endometrial lining of the ovarian cyst has extended through a perforation and has invaded the wall of the uterus, giving rise to the "adenomyoma" shown in Figures 57 and 58.

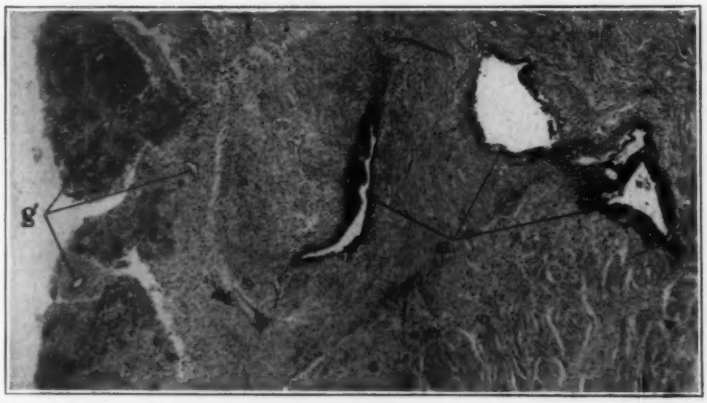

Fig. 61 (Case 5).—Another portion of the wall of the ovarian cyst which is fused with the uterus; ×25. Here the lining of the cyst (at the left) is replaced by an adenocarcinoma; an occasional histologically normal-appearing uterine gland is present (*g*). The benign endometrial invasion of the uterine wall is shown at *e*.

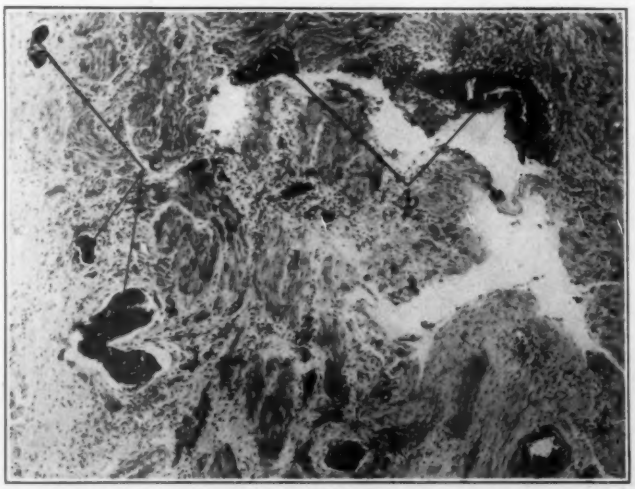

Fig. 62 (Case 5).—Another portion of the uterine wall just beneath the ovarian cyst. Both a benign (*b*) and a cancerous (*c*) "endometrial" invasion are present in the same field of the microscope. I was unable to demonstrate that the cancer arose from malignant changes in the endometrial tissue in the uterine wall, but I believe that both the benign and malignant tissue invaded the uterus from the ovary.

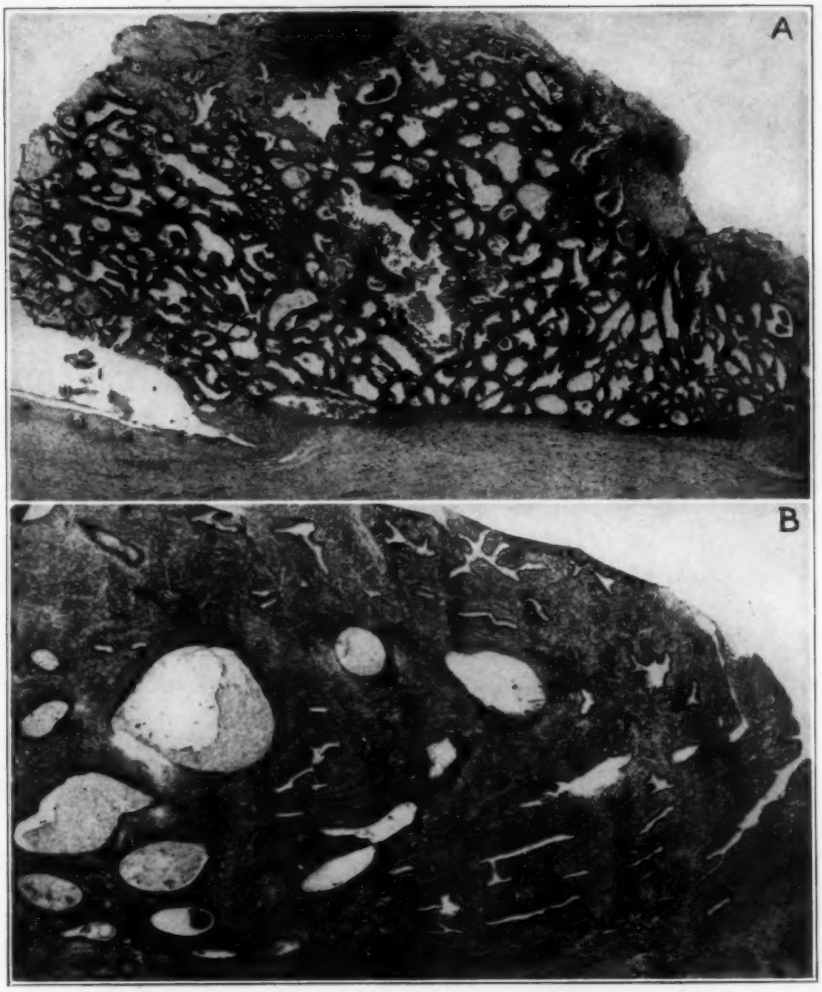

Fig. 63 (Case 5).—Two photomicrographs (× 25), one (*A*) of a malignant "endometrial" polyp projecting into the cavity of the ovarian cyst, and the other (*B*) of a benign polypoid hypertrophy of a portion of the mucosa lining the uterine cavity (same case). Malignant growths often reproduce the general structure of the tissue from which they primarily arose.

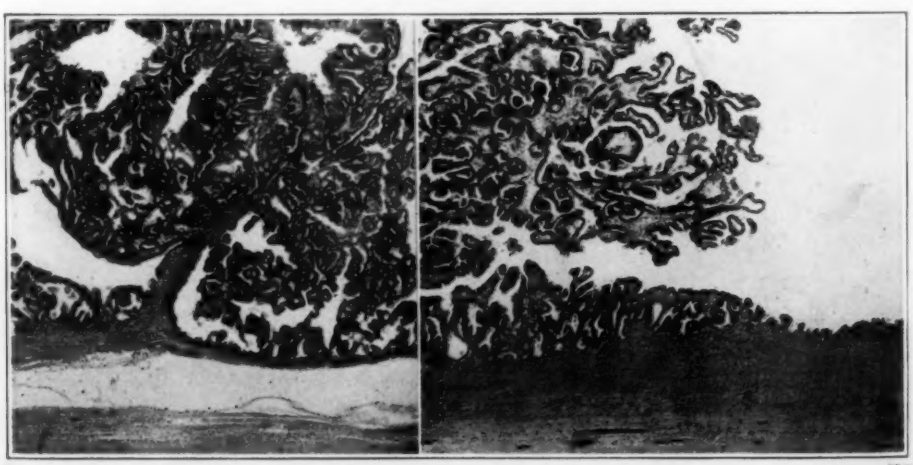

Fig. 64 (Case 5).—Different portions of the wall of a small cyst in the wall of the large one (Fig. 57); reduced one-sixth from photomicrograph with a magnification of 25 diameters. The gradual transformation of an almost histologically benign epithelial (endometrial) lining (right) into typical papillary endometrial adenocarcinoma (left) is illustrated.

present as an ovarian carcinoma probably arising in a benign endometrial cyst or hematoma. Through the courtesy of Dr. Elting I have had the opportunity to study the specimen. At the operation (Oct. 23, 1923), the wall of the cyst was found to be fused with the posterior surface of the uterus. There was no gross evidence of malignant implantations. The uterus, both tubes and ovaries and appendix were removed. Both tubes were attached to the walls of the cyst and appeared patent and normal. The uterus was slightly enlarged and contained multiple small intramural leiomyomas and an "adenomyoma" of its posterior wall arising from its posterior surface (Figs. 57 and 58). The uterine mucosa was hypertrophied and histologically benign (Fig. 63). The cyst apparently arose from the left ovary (I was unable to find the right ovary).

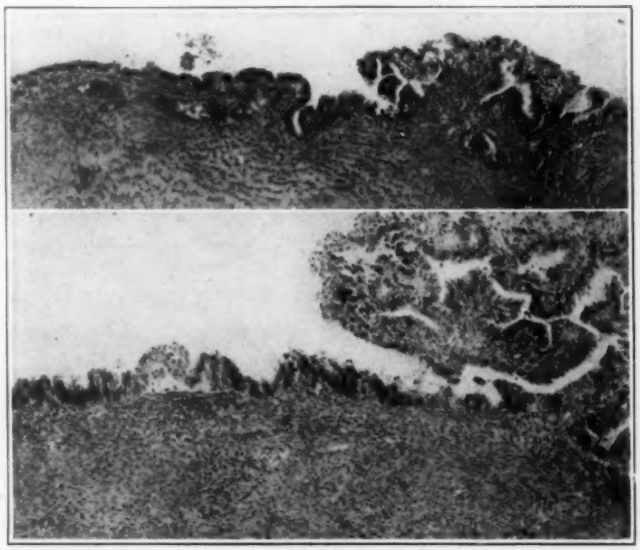

Fig. 65 (Case 5).—Different portions of the wall of the large endometrial ovarian cyst with carcinoma arising in its epithelial lining; ×60. The epithelial lining (left half of upper photomicrograph) is histologically benign in places, and similar to that found in portions of many benign endometrial cysts. At the right, this epithelium shows a hyperplasia and a resultant development of glands not definitely malignant. The epithelial lining of the left half of the lower photomicrograph shows both a hyperplasia and a metaplasia, and it is continuous with the epithelial lining of a definite adenocarcinoma (right), the latter having the same histologic structure as that found in the body of the uterus. The ovarian cyst is, in part, lined with typical benign endometrial tissue, with evidence of previous perforations and fusion of the cyst wall at their sites with the posterior uterine wall together with subsequent invasion of the uterine wall by the endometrial lining of the cyst, causing a typical benign "adenomyoma" of the uterus, as often occurs in these cases. The development of malignant changes in the epithelial lining of the cyst giving rise to an adenocarcinoma histologically identical with that often found in adenocarcinoma of the body of the uterus indicates that the ovarian carcinoma is an endometrial carcinoma arising in the lining of a typical benign endometrial ovarian cyst or hematoma.

Papillary masses of the malignant growth projected into the cavity of the cyst, and in places its wall was invaded by a similar growth. In other situations, the histologic structure of the lining of the cyst was identical with that often found in benign endometrial cysts, namely, typical uterine mucosa in some situations and in others a single layer of cuboidal and columnar epithelium, and, in places where the epithelium was lacking, phagocytic cells and blood pigment similar to that shown in Figures 26 and 55. The carcinoma arose in the benign endometrial lining of the cyst. The most interesting portion of the cyst wall was that which was fused with the uterine wall. Here, benign endometrial tissue was found extending through breaks in the wall of the cyst (possibly where perforation had occurred), and it had invaded the wall of the uterus, giving rise to the adenomyoma already described (Figs. 59 and 60). Malignant tissue was also found invading the posterior uterine wall (Fig. 62). The patient had a satisfactory convalescence.

Interpretation of Findings.—My interpretation of the findings in this case is as follows: As a result of a back-flow of menstrual blood through the tubes into the peritoneal cavity, endometrial tissue became implanted on the surface of the ovaries and possibly other pelvic structures. A typical endometrial cyst or hematoma developed in the left ovary, which perforated, probably giving rise to secondary implantations. The wall of the ovarian cyst at the site of perforation became fused with the posterior uterine wall, a condition so often occurring in benign endometrial cysts of the ovary. The adenomyoma of the uterus arose from endometrial tissue escaping through the perforation. The carcinoma, of the same histologic structure as that arising in the body of the uterus, developed in the endometrial lining of the ovarian cyst.

CASE 6.—*Peritoneal carcinosis, most marked in the omentum and in the posterior cul-de-sac, carcinoma of both ovaries, more advanced in the right and replacing nearly all of the ovarian tissue; histologically benign endometrium-like tissue, present in the right ovary and cancer arising from or replacing its epithelium.*

History.—The patient, aged 53, complained of abdominal distention of only a few weeks' duration. She was married, but had never been pregnant. The menopause had occurred three years before. The abdomen was distended and presented the physical signs of ascites. The pelvic examination showed induration in the cul-de-sac, but otherwise was negative. The preoperative diagnosis was peritoneal carcinosis.

Operation.—The operation occurred at the Albany Hospital, July 22, 1922. When the abdominal cavity was opened, a large amount of fluid was found, together with extensive malignant peritoneal implantations. The omentum was extensively involved, especially along its attachment to the transverse colon. The gallbladder and the stomach were normal on palpation. Both ovaries were enlarged, and the right one was densely adherent to the posterior surface of the lower portion of the right broad ligament. The uterus, both tubes, the ovaries and appendix were removed. The patient made a satisfactory convalescence and before leaving the hospital was treated with the roentgen rays. She returned to the Albany Hospital in October, 1922. The fluid had reaccumulated, and the patient was very uncomfortable. The fluid was removed through a small abdominal incision under procain. A marked retrogression of the peritoneal implantations was noted, many of the smaller ones having disappeared. In the latter part of January, 1923, I advised her to consult Dr. C. F. Burnam, at Dr. Howard A. Kelly's Hospital, in Baltimore, Md., where she was treated

with radium. She was more comfortable for a few months after this treatment, but the fluid gradually reaccumulated, and the abdomen was aspirated in June, 1923. The patient died the latter part of August, 1923.

Both ovaries were slightly enlarged, the right one being the larger, by an adenocarcinoma invading and replacing the ovarian tissue (Fig. 66). This invasion apparently occurred from the lateral surface of the ovaries (Fig. 67). The uterus contained small intramural leiomyomas and a large benign cystic endometrial polyp, which had distended the uterine cavity. Benign endometrial implants were not found on the posterior surface of the uterus. The right tube

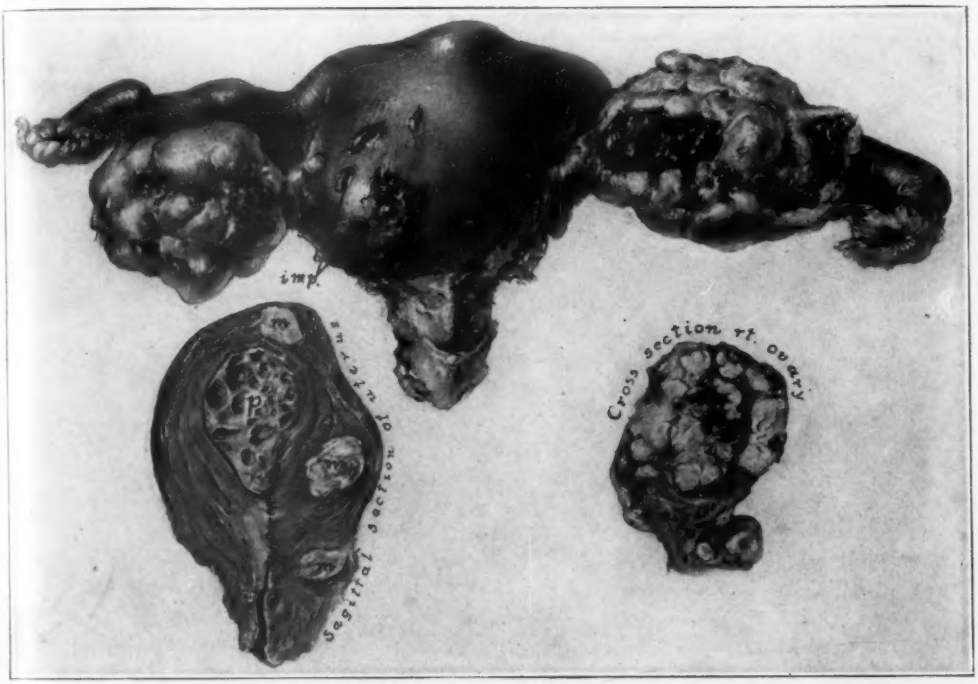

Fig. 66 (Case 6).—Posterior view of the uterus, tubes and ovaries; sagittal section of the uterus (showing a cystic polyp (*p*) and leiomyomas (*m*)), and a cross section of the right ovary and tube; × 5/6. The ovaries are enlarged (patient aged 53) and are both invaded by cancer, more advanced in the right one and replacing nearly all the ovarian tissue as shown in the cross section of the ovary (Fig. 67). Benign peritoneal endometrial implants were not found. If they had been present in the cul-de-sac (their most frequent situation), they would have been covered by the extensive implantation of cancer. Both tubes were apparently patent, but the fimbriae of the left one were invaded by cancer implanted on their peritoneal surface. The histologic structure of a malignant peritoneal implant on the posterior surface of the uterus, may be seen in Figure 8.

was apparently normal and patent, and cancer cells were not found in its lumen. The left tube was patent and cancer cells were not found in its lumen, but the tissues of its fimbriated extremity were invaded by cancer apparently implanted

on its peritoneal surface. The only evidence found of benign endometrium-like tissue in the ovaries was in a small cyst (Fig. 68) on the lateral surface of the right ovary, which was lined by cuboidal and columnar epithelium (a few of which were ciliated). In places, this epithelium showed a hyperplasia with the formation of papillary projections into the cavity of the cyst, and also a metaplasia gradually assuming a malignant structure. This cyst could be of implantation origin, although its origin from the surface epithelium cannot be excluded.

Interpretation of Findings.—The following interpretations of the findings in this case may be made. Implantation endometrium-like tissue was present in the

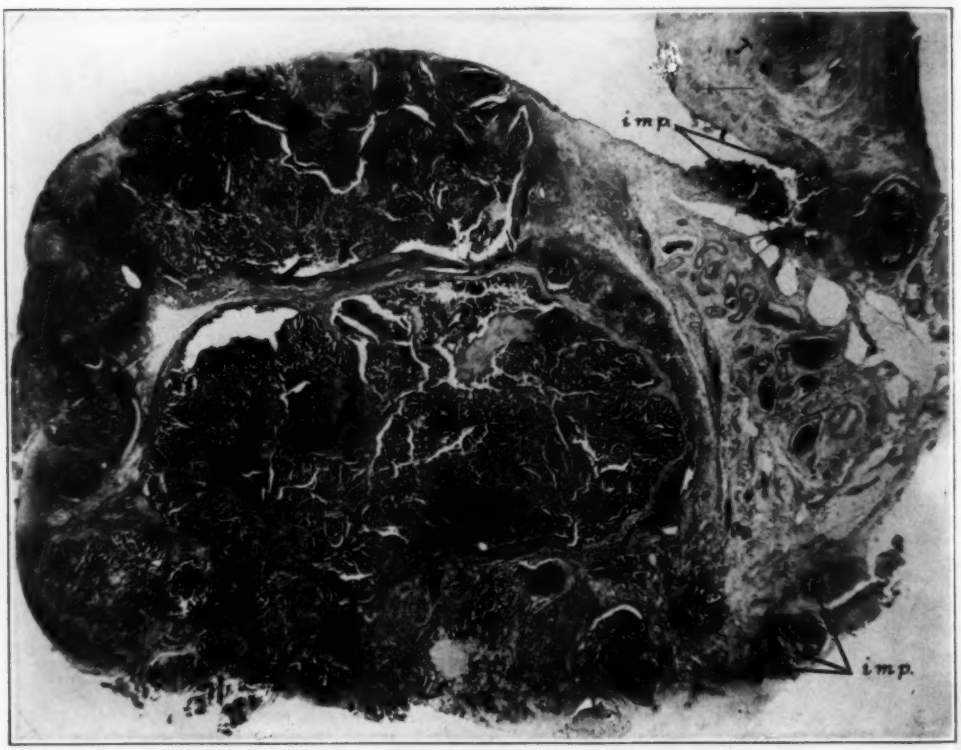

Fig. 67.—A cross section of the right ovary; × 5. The ovarian tissue is almost entirely replaced by an adenocarcinoma of a type often found in the body of the uterus. This carcinoma apparently primarily invaded the ovary from its lateral surface, the portion of the ovary most frequently involved in benign endometrial implantations. Histologically, benign endometrium-like structures were found in this situation (Fig. 68). Malignant implants (*imp.*) are present, invading the broad ligament. Malignant changes in ovaries containing a small amount of endometrial tissue (Fig. 69) could readily cause a condition similar to that found in this case.

right ovary, and possibly also in the left one and in the cul-de-sac. The implantations were few and had remained dormant, a condition that occurs in a large percentage of these cases. Adenocarcinoma developed in benign endometrial

tissue on or near the lateral surface of the right ovary, possibly similar to the endometrial tissue shown in Figure 69. It invaded the tissues of the ovary and, as it was situated on or near its surface, peritoneal implantations occurred early. The left ovarian carcinoma may have arisen primarily from benign endometrial tissue in it, or by implantation from the right ovary just as the peritoneal implantations arose. Any benign peritoneal implantations possibly present in the cul-de-sac had been covered up by the extensive malignant implantations in that situation. The reason for this interpretation is as follows: Histologically, it was an adenocarcinoma of a type frequently found in cancer of the body of the uterus. It apparently developed primarily on or near the lateral

Fig. 68 (Case 6).—A small cyst of the endometrial type near the lateral surface of the right ovary; ×25. The histologic structure of portions of this cyst is similar to that shown in Figures 15 and 34. The epithelial lining is, for the most part, columnar, and a few ciliated cells were found. It is of a type which could have arisen either from an endometrial implantation or from the true surface epithelium of the ovary. There is a hyperplasia and also a metaplasia of the epithelial lining, with the formation of papillae in places quite similar to that shown in Figure 29. In other sections of the same cyst these changes in the epithelial lining resemble malignancy. An adenocarcinoma of a type often present in the body of the uterus was found, apparently primarily invading the ovary from its lateral surface and associated with histologically benign tissue of an endometrial type, which in places showed possible early malignant changes. These facts suggest that the ovarian carcinoma could be an endometrial carcinoma arising in endometrial tissue in the ovary, which was small in amount and was associated with a small number of peritoneal implants or none. This represents the largest group of cases with endometrial implantation in the ovary (Figs. 12 and 69), and the most difficult one to recognize, especially after malignancy has developed, as the malignant changes in the endometrial tissue in the ovary may replace all of this tissue, and malignant peritoneal implants may cover any benign endometrial implants possibly present.

surface of the right ovary, the very situation in which benign endometrial implantation most frequently occurs. A small "endometrial" cyst, lined in part by histologically benign "endometrial" epithelium, was present in the ovary near its lateral surface (compare Fig. 15). Malignant changes had occurred in portions of this cyst. The largest groups of cases with benign endometrial implants are those with a few implants, usually in one or both ovaries and in the peritoneum, which remain dormant. Cancer developing in the ovarian endometrial tissue of a typical case (Fig. 69) of this group would very readily give rise to the conditions found in this case.

CASE 7.—*Peritoneal carcinosis, most marked about and invading the sigmoid, in the omentum and posterior and anterior cul-de-sac; implantations on the mesial surface of both ovaries, larger and more numerous on the left; benign endometrial invasion of the left side of the posterior uterine wall and the left ovary; the "endometrial" glands of the left ovary were situated about the base of the largest "implantation," which had the structure of a malignant papilloma, and cancer was found apparently replacing or arising from these glands (the possibility of the cancer on the surface of the ovaries arising from implantations from carcinoma due to malignant changes in endometrial tissue on the surface of the sigmoid must also be considered).*

History.—The patient, aged 54, was first seen Dec. 16, 1922. Her chief complaint was constipation and indefinite pains in the right side of the abdomen. She had had two children, the last one twenty-one years before. The menopause had occurred at the age of 46. The patient had been constipated for several years, and it was impossible definitely to determine the duration of the present illness.

Examination.—The patient was well nourished, and the result of abdominal palpation was negative. The pelvic examination also was negative; no induration could be felt in the cul-de-sac. The urine was normal. Roentgenograms taken of the gallbladder region and of the gastro-intestinal tract were negative. No diagnosis was made. The patient was not seen again until July, 1923. She stated that she had felt a little better for a few months after the previous examination, but that the constipation had increased and that recently her abdomen had seemed larger. The abdomen was distended and presented the physical signs of ascites. Pelvic examination showed induration in the cul-de-sac, but otherwise was negative. A preoperative diagnosis ot peritoneal carcinosis was made. The operation occurred at the Albany Hospital July 12, 1923. When the peritoneal cavity was opened, a large amount of fluid was found together with extensive malignant peritoneal implantations and involvement of the omentum. The gallbladder and stomach were normal on palpation. The sigmoid was adherent at the pelvic brim and the left side of the pelvis. The wall of the sigmoid was invaded from its peritoneal surface by the growth, thus narrowing its lumen and causing almost complete obstruction such as was indicated by the distention of the sigmoid above. The uterus, both tubes and ovaries, and the appendix, which was studded with implants, were removed. A loop of the sigmoid was brought into the abdominal wall on the left side and a rubber tube sutured into its lumen. The patient recovered from the operation, but died three months later. The relatives of the patient refused a postmortem examination. Radium and roentgen-ray therapy were not employed, as apparently they had been ineffective in two previous cases in which they had been used.

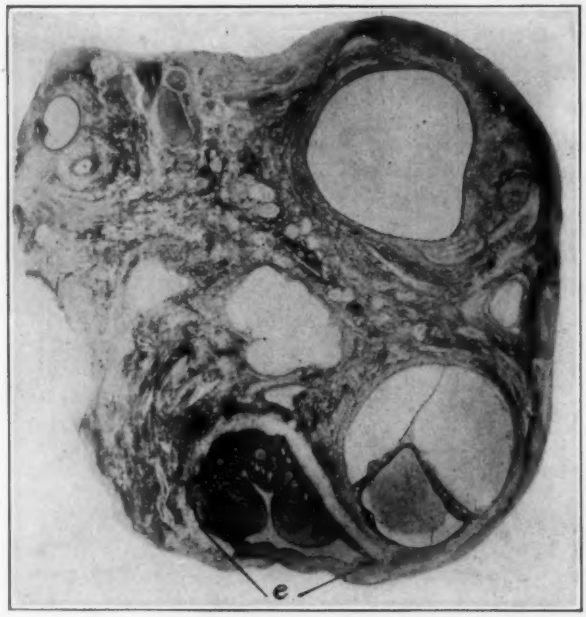

Fig. 69.—A cross section of an ovary with a small amount of benign endometrial tissue (*e*) imbedded in it near its lateral surface; × 5. Similar tissue was also present in the opposite ovary, and only one peritoneal implant was found, which was in the cul-de-sac. Carcinoma arising in the endometrial tissue of the ovaries in a case similar to this one could give rise to the lesions shown in Case 6.

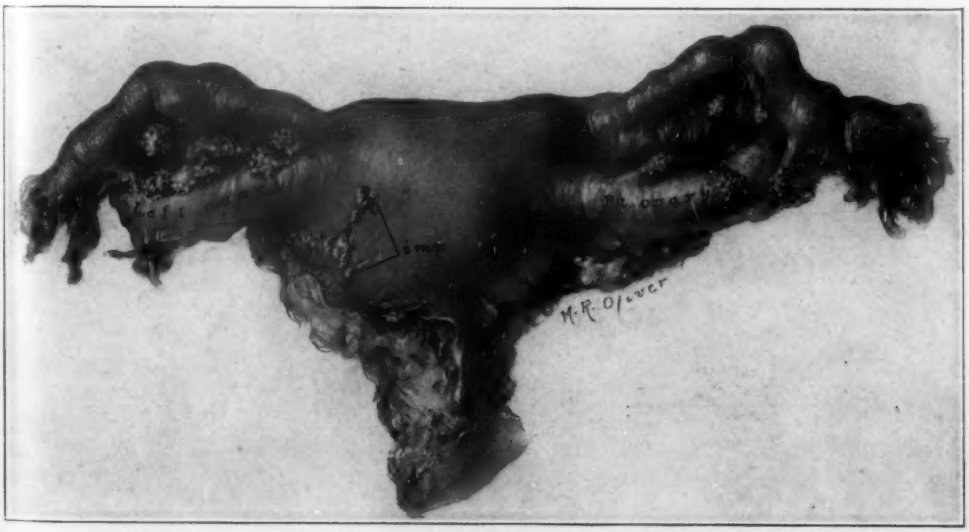

Fig. 70 (Case 7).—Posterior surface of the uterus, tubes and ovaries (× 5/6). The posterior surface of the uterus appears normal except for the presence of a benign implantation, endometrial invasion on the left side, associated with small malignant implants (*imp.*) The tubes are apparently patent and normal. Malignant implants are present on the posterior surface of both mesosalpinges and the upper and latter surfaces of the atrophic ovaries (patient aged 54). These were larger and more numerous on the left ovary. One implantation on the left ovary differed from the others. It was larger, appeared older, and had the structure of a malignant papilloma (Fig. 73).

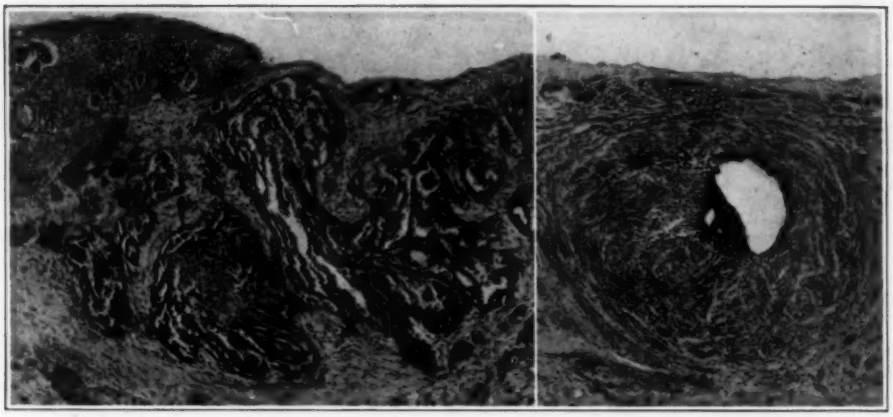

Fig. 71 (Case 7).—Different portions of the same section of the posterior uterine wall, from the area indicated by *imp.* in Figure 70; × 60. Malignant implantation invasion (left), both a recent one on the surface, at the left, and an older one invading the deeper tissues of the uterus is shown. The section at the right could be interpreted as a circumscribed endometrial leiomyoma resulting from a benign implantation endometrial invasion, as shown in Figure 4. A normal-sized gland and a dilated gland of endometrial type are present, with tissue about them resembling endometrial stroma, and an occasional ciliated cell was found in the dilated gland.

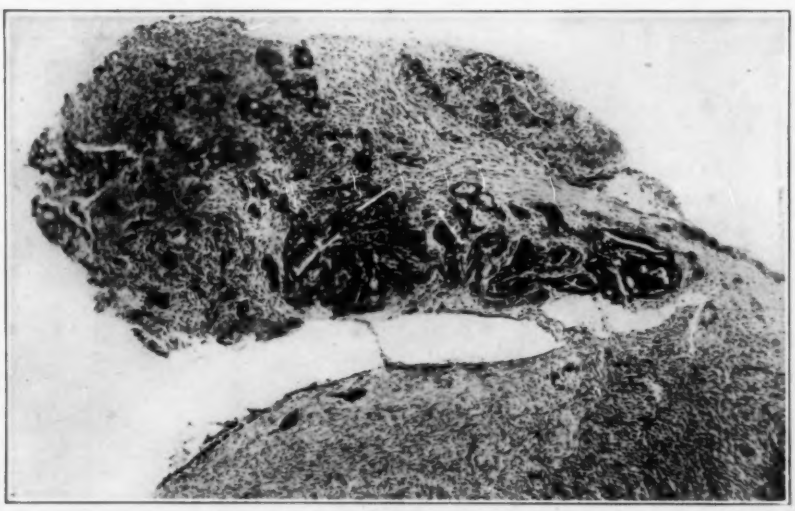

Fig. 72 (Case 7).—A malignant implant on the surface of the left ovary, showing the reaction of the ovarian tissue against the implantation; × 25. The ovarian tissue has grown out into the implant forming a polypoid growth.

The uterus was normal in size. Benign endometrial tissue was found in two areas of the posterior uterine wall just beneath the peritoneum near the attachment of the left broad ligament (Fig. 71). A small benign polyp was present in the uterus. The rest of the uterine mucosa was atrophic. Both tubes appeared normal, and bits of cancer were not found in their lumen. The right ovary was atrophic. On its surface small implants of cancer were present, which were similar to those found on the peritoneum. Similar implants were also present on the surface of the left ovary, and, in addition, one was found larger and more definitely papillomatous than the others. This malignant

Fig. 73 (Case 7).—The largest and apparently the oldest implant on the surface of the left ovary; × 25. Possibly it represents a later stage of the condition shown in Figure 72. It has the structure of a papilloma similar to that shown in Figure 33. Its pedicle is vascular, and about its base are three glands (*e*) of endometrial type (although their origin from the surface epithelium of the ovary cannot be excluded), and of the same structure as the glands in the uterine wall shown in Figures 1 and 2, except that there was not any tissue about them resembling endometrial stroma. An occasional ciliated cell was present. At *c* a gland-like space is present, lined in part by histologically benign epithelium and presenting the picture of cancer arising in or invading it. The adenocarcinoma is of a type sometimes found in cancer of the body of the uterus. The primary growth may be the papilloma just described, which may have developed in endometrial structures implanted on the surface of the ovary or from the true surface epithelium of the ovary, as indicated in this photomicrograph as well as in Figures 34 and 35. Implantations would occur early, and the lateral surface of the sigmoid, on account of its proximity to the left ovary, would naturally be extensively involved. Another interpretation presents itself; namely, that the primary malignant growth was elsewhere than in the ovary. It is possible that it may have arisen in malignant changes in endometrial tissue implanted on the sigmoid, which was the seat of the most extensive and apparently the oldest growth.

papilloma was about 5 mm. in its greatest diameter and was attached to the ovary by a vascular pedicle (Fig. 73). It resembled, in its gross appearance, the papilloma shown in Fig. 33 and may have had a similar origin. Glands of a benign endometrial type with an occasional ciliated cell (similar to those shown in Figure 23, which, as in the case of the latter, may have arisen from the true surface epithelium of the ovary) were present in the ovary about the base of the pedicle of the papilloma, and cancer was found, apparently arising in these or replacing their epithelium. The type of cancer was similar to that sometimes found in adenocarcinoma of the body of the uterus. Ciliated epithelium was found in the tubal mucosa, in the endometrial glands of the posterior uterine wall and in the glands of the ovary about the base of the papilloma. None were found in the uterine mucosa.

Interpretation of Findings.—My interpretation of the findings in this case is as follows: Sometime during the menstrual life of the patient, blood escaped through the tubes into the peritoneal cavity carrying with it bits of uterine mucosa (possibly in this instance tubal epithelium, since the epithelium lining these glands resembled that of the tube more than that of the uterus). Implantations occurred on the left side of the posterior surface of the uterus, on the left ovary and possibly on other pelvic structures, including the sigmoid. Subsequently, malignant changes occurred in the ovarian implant giving rise to a malignant papilloma. Since it was situated on the surface of the ovary, secondary implantation occurred soon after the development of the papilloma, the most extensive and apparently oldest of which were found on the lateral surface of the sigmoid, which was in close proximity to the left ovary. Another interpretation presents itself. The malignant changes may have occurred in endometrial tissue implanted on the surface of the sigmoid. The malignant peritoneal implantations and those on the ovaries resulted from this. The majority of the peritoneal and ovarian implants were sessile and invasive, but a few were present with a tendency to form papillary outgrowths resembling the papilloma that was considered as the primary malignant tumor.

THE CLASSIFICATION AND FREQUENCY OF ENDOMETRIAL CARCINOMA OF THE OVARY

Patients with benign endometrial tissue in the ovary or ovaries may be divided into two main groups. In the smaller and more easily recognized group, a hematoma or cyst has developed in one or both ovaries (Figs. 28 and 33). Such ovaries are usually adherent to the posterior surface of the uterus, broad ligament, or side of the pelvis as a result of perforation of the hematoma or from endometrial tissue on the surface of the ovary invading those structures, thus fusing the ovary with them. These cases are nearly always associated with extensive peritoneal implantations and an endometrial invasion of the posterior surface of the uterus. If cancer should develop in the epithelial lining of an endometrial cyst or hematoma, it would remain within the cyst until it had extended through its walls or perforation had occurred. With our knowledge of benign endometrial cysts or hematomas and their associated lesions, it is possible to recognize at

operation that the cancer has developed in, or is associated with, a benign endometrial cyst or hematoma. Benign endometrial tissue may be readily found in the histologic examination of sections from the wall of the cyst and other pelvic structures, especially in the examination of sections from the posterior wall of the uterus, if the sections are taken from likely situations such as are shown in Cases 4 and 5.

In the group that is larger and more difficult to recognize, ovarian hematomas have not developed, and the only ectopic endometrial tissue may be that in the ovary or ovaries, but usually peritoneal implants are also present, especially in the cul-de-sac and on the posterior surface of the uterus and broad ligaments (Figs. 11 and 12). These implants are usually few in number and of small size and are only occasionally as extensive as those generally associated with an ovarian hematoma. The endometrial tissue in the ovary is also usually small in amount and consists of endometrial structures similar to those found in the invasion of the uterine wall by the mucosa lining its cavity. In some instances glands, normal-sized or dilated, are present with or without endometrial stroma about them. If the latter is present, it is usually small in amount (Figs. 10 and 15). In other instances, small masses of histologically typical uterine mucosa are present (Figs. 3, 6 and 69). In still other specimens, all of these endometrial structures occur. The endometrial tissue is most frequently situated on or near the lateral and under surfaces of the ovary (Figs. 13, 14 and 69). Should malignant change arise in the endometrial tissue of this group, it would develop on or near the surface of the ovary, invading the tissues of that organ forming a solid tumor or possibly occasionally a malignant cyst. Malignant implantation would occur early, easily obscuring or covering any benign peritoneal implantation that might be present. The small amount of benign endometrial tissue in the ovary would soon be obscured or replaced by the cancer. These factors would make it difficult to recognize any evidence of benign endometrial implants at the time of the operation or in the histologic study of the specimen removed; this is well shown in Cases 6 and 7.

Ovarian carcinoma of endometrial origin may be classified as follows (this is only a working classification, as it is based on so few cases):

1. Carcinoma arising in endometrial tissue in the ovary.

(*a*) Carcinoma arising in a small benign endometrial implant on or near the surface of the ovary. One would infer that these cancers would usually not be cystic.

(*b*) Carcinoma arising in the epithelial lining of a benign endometrial cyst or hematoma. One would infer that these cancers would be cystic.

2. Implantation carcinoma of the ovary from malignant tissue escaping through the tubes from a primary growth of the mucosa of the body of the uterus as stated by Graves [8] in his classification of ovarian tumors and as has been described by me.[4]

3. Implantation carcinoma of the ovary from cancer arising in a peritoneal endometrial implant.

4. Carcinoma metastatic through the lymph vessels or veins from cancer of the body of the uterus.

It is a question whether or not endometrial tissue can arise from the true surface epithelium of the ovary before and after birth. If so, cancer might arise in this.

I have studied nineteen specimens of ovarian carcinoma endeavoring to ascertain whether or not the carcinoma could have been of endometrial origin with the following results.

In three specimens histologically benign endometrium-like tissue was found on or near the surface of the ovary and cancer was found replacing its epithelium or arising from it.

In four specimens (Cases 2, 3, 4 and 5), evidence of what could be interpreted as a previously benign ovarian cyst or hematoma of endometrial origin was found, and the cancer apparently arose in its epithelial lining or was replacing it from some other source. It bore the same histologic relation to the benign endometrial tissue in the specimen that cancer of the body of the uterus bears to the benign portions of the uterine mucosa adjacent to the growth (for a further discussion of the origin of the cancer in these specimens see report of cases and illustrations).

Three instances of malignant ovarian tumors were found associated with cancer of the body of the uterus, the ovarian tumors apparently being secondary to the uterine, just as benign endometrial implants arise in the ovary from bits of uterine mucosa escaping through the tubes. In one of these cases, apparently both an endometrial sarcoma and carcinoma were present in the uterus, the implantations being sarcomatous in type. These three cases have been reported in a previous communication.[4] In six of the other nine specimens, three solid and three cystic, the histologic structure of the carcinoma was similar to that found in adenocarcinoma of the body of the uterus, but no evidence of benign endometrial implantation was found. The carcinoma could easily have replaced all of the benign endometrial tissue in the ovary or ovaries, and the extensive malignant peritoneal implantations present in each case could have obscured or covered any benign peritoneal endometrial implants present.

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8. Graves, W. P.: Gynecology, Philadelphia, W. B. Saunders Company, 1923, p. 404.

In the remaining three specimens, the ovarian tumors were large (bilateral in two instances), of the solid type and different in appearance from the others, although two of them were definitely adenocarcinoma. There was no evidence of benign or malignant peritoneal implantations except in one, and benign endometrial tissue was not found in the ovaries. In one, a typical benign endometrial implant was present on one of the tubes.

One instance was found of possible malignant implantation on the ovary, secondary to a carcinoma on the surface of the sigmoid, and possibly due to what might be interpreted as malignant changes in a benign peritoneal endometrial implant (Case 6). I do not believe that all of the malignant epithelial tumors of the ovary are of endometrial origin, but some are and possibly the percentage is large. This is in accord both with the frequency of misplaced endometrial tissue in this organ and also with the endometrial type of many ovarian carcinomas. A very important suggestion presents itself, namely, that malignant changes possibly occur with greater frequency in ectopic endometrial tissue in the ovary than in ectopic endometrial tissue in other structures of the pelvis and even with greater frequency than in the mucosa of the body of the uterus.

THE CLINICAL SIGNIFICANCE OF MALIGNANT ENDOMETRIAL TUMORS OF THE OVARY

I shall not discuss the treatment of ovarian carcinoma except as the result of these studies may possibly bear upon it. It is necessary to appreciate the following facts:

1. Carcinoma arising in endometrial tissue on or near the surface of the ovary, similar to that shown in Figures 3, 6, 13, 14 and 69, would early give rise to implantation and the chance of operating on a person with such a condition before this had occurred would be small.

2. Carcinoma arising in the epithelial lining of an ovarian endometrial cyst or hematoma (Figs. 28 and 33) would not give rise to implantation unless it had extended through the wall of the cyst or perforation had occurred. Furthermore, the cyst is usually adherent to the posterior surface of the uterus, broad ligament or side of the pelvis as a result of a previous perforation of the benign ovarian cyst or as a result of benign endometrial tissue on the surface of the ovary (Figs. 52 and 57). If the cyst were freed from the structures adherent to it, rupture would occur with the probable dissemination of bits of cancer throughout the peritoneal cavity. If possible, these cysts should be removed intact, together with the structures adherent to them, such as the uterus or the posterior layer of the broad ligament. If this is not possible, the cyst should be completely aspirated before it is freed and measures taken to prevent the contamination of the peritoneal cavity with its contents.

3. Apparent malignant implants in the cul-de-sac of a patient with a malignant ovarian cyst may be benign endometrial implants, associated with an ovary containing endometrial tissue in which the cancer may have developed, a condition revealed in Cases 2 and 3.

4. If benign endometrial tissue is present in one ovary, it is frequently present in the other also. Malignant ovarian tumors are likewise frequently bilateral. In a certain percentage of the cases there is an associated carcinoma of the body of the uterus. The uterine wall may also be directly invaded by the ovarian cancer adherent to the latter or by metastasis. It is wiser, therefore, to remove both ovaries and the uterus in operating on patients with ovarian carcinoma.

5. If it should be demonstrated that cancer is more apt to arise in endometrial tissue in the ovary than in the mucosa of the uterus, this endometrial tissue in the ovary must be considered as potentially malignant. This particular phase of the subject needs careful investigation in order that we may more intelligently treat patients harboring benign endometrial tissue in the ovary.

CONCLUSION

Endometrial tissue in the ovary is of common occurrence. The evidence obtained [4] would indicate that it usually develops from the implantation of bits of uterine mucosa escaping through the tubes during a retrograde menstrual flow from the uterine cavity. Tubal epithelium also may possibly become implanted on the ovary. It is difficult to disprove that endometrium-like tissue may not occasionally arise from the true surface epithelium of the ovary; I have seen a few specimens which might be so interpreted.

The evidence in favor of malignant changes in endometrial tissue in the ovary as a source of some, and possibly of many, ovarian carcinomas may be summarized as follows:

1. Endometrial tissue in the ovary is of frequent occurrence, having been found present in one or both ovaries in 64 of 332 abdominal operations for pelvic conditions this last year.

2. It is natural to assume that malignant changes should occur in it, as it is governed by the same natural laws as the uterine mucosa, in its reaction to menstruation, pregnancy, and the menopause, and the latter becomes malignant. In addition to at least the same potentiality for malignant changes as the uterine mucosa, there is the contributing factor of its being ectopic.

3. One of the most striking features of malignant ovarian tumors and their associated peritoneal implants is their close resemblance to benign endometrial lesions of the ovaries, especially to perforated cysts or hematomas and their associated benign endometrial implants.

4. Many adenocarcinomas of the ovary have the same histologic structure as adenocarcinoma of the body of the uterus, namely, endometrial carcinoma.

5. Malignant ovarian tumors are frequently bilateral, as are also benign endometrial growths in the ovary.

6. Bits of cancer from cancer of the body of the uterus may escape through the tubes, causing implants on the peritoneum and the surface of the ovaries; the latter may develop into ovarian carcinomas.

7. The age incidence of certain types of ovarian cancers corresponds with that of cancer of the body of the uterus.

8. Ovarian papilloma (which frequently becomes cancerous) has been reported arising from the fimbriae of the tube.[7]

9. This acquired endometrial tissue in the ovary, which is of common occurrence, offers in part the most satisfactory explanation for the frequency of ovarian carcinoma as compared with that of the testis, both organs having a common embryonic origin.

10. In seven of sixteen specimens of ovarian carcinoma studied (without any evidence of cancer of the body of the uterus), benign endometrium-like tissue in the ovary was found, bearing the same histologic relation to the malignant growth that the benign uterine mucosa bears to the adenocarcinoma arising in the body of the uterus. The carcinoma had either arisen in this tissue or was invading it from another source. In all seven instances, the histologic structure of the carcinoma was similar to that found in some cancers of the body of the uterus.

In four of these seven cases, the carcinoma could be interpreted as having developed in the epithelial lining of a benign cyst of endometrial origin which had become fused with other pelvic structures at the site of a previous perforation of the cyst, and was associated with a benign endometrial invasion of the posterior uterine wall. In two of these four cases other interpretations could be made.

A brief summary of the evidence in these four cases follows:

In Case 5, the cancer arose in the epithelial lining of an easily recognized benign endometrial cyst.

In Case 4, there was a larger malignant cyst whose cavity communicated with that of a smaller one. The smaller cyst was in part lined by benign endometrial tissue, and cancer was replacing or arising in this tissue. I believe that the larger cyst was likewise primarily an endometrial cyst, not only because it communicated with the smaller one, but also because the nonmalignant portion of its wall was identical with that often present in large endometrial cysts where the greater portion of the epithelial lining has been cast off.

In Case 3, the cancer had developed in the epithelial lining of an apparent serous papillary cystadenoma. The evidence present in the specimen (see report of case and illustrations) suggests that the cyst could have been of implantation origin.

In Case 2, the endometrial tissue present in the wall of the malignant cyst bore such an intimate relation to the epithelial lining of the cyst as to suggest that the cyst might have arisen primarily in this tissue. The carcinoma bore the same histologic relation to portions of this endometrial tissue that one finds in cancer of the body of the uterus. On the other hand, the gross appearance of the cyst was that of a pseudomucinous cystadenoma, and the histologic structure of portions of the cyst would suggest that the carcinoma may have arisen in the epithelial lining of such a cyst and that it had secondarily invaded the benign endometrial tissue in its walls.

In the other three of the seven cases, the carcinoma may have arisen in endometrial tissue in the ovary, which had not developed into an endometrial cyst.

In the nine remaining carcinomas, endometrial tissue was not found, although it could have been present in some of these. The malignant growth may have replaced all the benign endometrial tissue present in the ovary, which may have been small in amount, and the associated malignant implants may have covered any benign peritoneal implants present.

I do not believe that all carcinomas of the ovary are of endometrial origin. Some are, however, and possibly the percentage is large, a fact that is in accord both with the frequency of endometrial tissue in that organ and with the endometrial type of many ovarian carcinomas.

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POSTOPERATIVE MASSIVE COLLAPSE OF THE LUNG *

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I. INTRODUCTION

For the conscientious surgeon, the end of the operation is only the beginning of a period of special attention to the patient. Pulmonary complications form one of the important problems of this time. Excellent analyses of such complications have appeared in recent years from several clinics.[1] Of all patients operated on, about 3 per cent. develop some type of pulmonary complication, and one of every 200 operations results fatally from this cause. These facts alone suffice to demonstrate the importance of this subject.

There are unquestionably several etiologic factors responsible for these postoperative pulmonary conditions, and those interested in the subject have divided the resultant complications into various clinical groups. Many of these groups have been carefully studied, but in spite of the general interest in postoperative pulmonary complications as a whole, there remains a special group of cases, classified as massive collapse of the lung, which has received but slight attention. The study of this complication is of importance in two ways: (1) as a clinical syndrome, and (2) as a mechanism in postoperative pulmonary complications.

1. It presents itself as a definite clinical entity which has escaped recognition in most instances. The only standard textbook found to contain an adequate description of this syndrome was one of the systems of medicine.[2] Some recent surgical treatises [3] briefly refer to it as a complication of gunshot wounds, but do not treat it as a postoperative complication.

2. The mechanism of collapse probably plays an important rôle throughout the field of pulmonary complications. Elwyn [4] comes to the

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* From the surgical clinic of Dr. Harvey Cushing, the Peter Bent Brigham Hospital.

1. Cutler, E. C., and Hunt, A. M.: Postoperative Pulmonary Complications, Arch. Surg. **1**:114 (July) 1920; Arch. Int. Med. **29**:449 (April) 1922. Whipple, A. O.: A Study of Postoperative Pneumonitis, Surg., Gynec. & Obst. **26**:29 (Jan.) 1918.

2. Bradford, J. R.: Massive Collapse of the Lungs, Oxford Medicine **2**:127, 1920.

3. Heuer, G. J.: Surgery of the Thorax in Keen's Surgery **8**:351, 1921. Choyce, C. C.: A System of Surgery, New York, **3**:362, 1923.

4. Elwyn, H.: Postoperative Pneumonia, J. A. M. A. **82**:384 (Feb. 2) 1924.

conclusion that most cases of postoperative pneumonia arise in atelectatic areas. Lee,[5] in a recent article, asserts his belief that collapse of the lung is a constant factor in all postoperative pulmonary complications. The present study, therefore, is divided into two parts. The

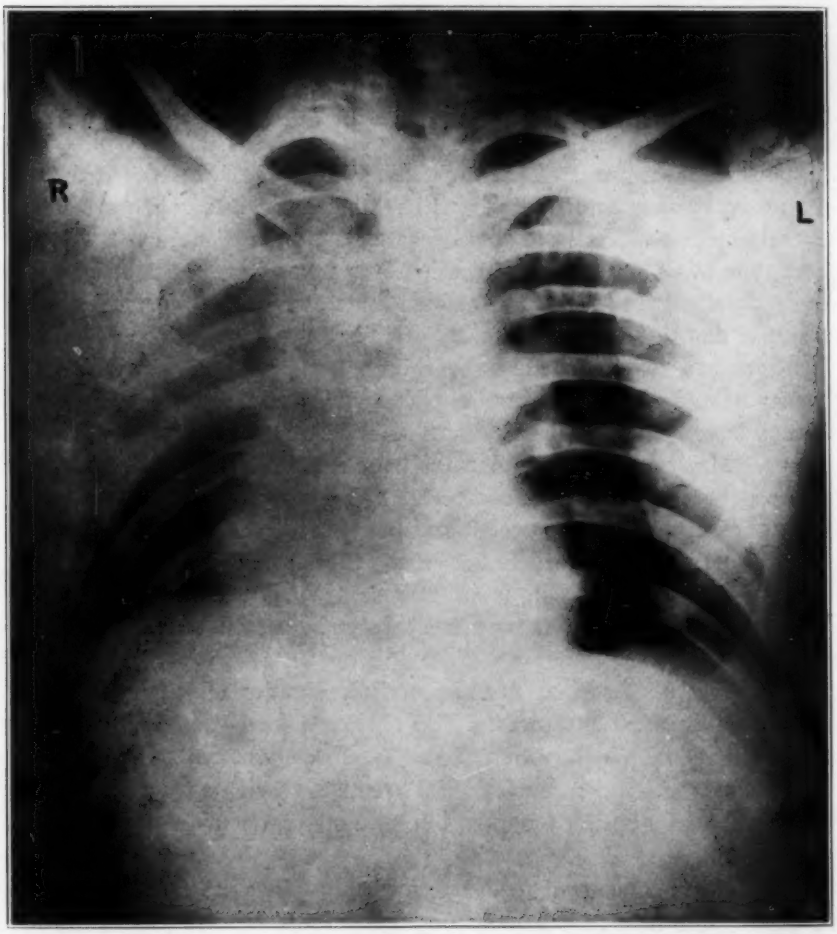

Fig. 1 (Case 38).—Postoperative massive atelectasis, right, the day following operation when physical signs were just developing, and temperature and respiration were rising sharply; the heart and trachea are obviously displaced. It should be noted in particular that the right base is still clear.

first concerns the clinical aspects of postoperative massive collapse, and includes not only a report of four typical cases, but also a compilation

5. Lee, W. E.: Postoperative Pulmonary Complications, Ann. Surg. **79**:506 (April) 1924.

and analysis of all the cases in the literature that have been fully recorded. The second part treats of the mechanism responsible for the condition and its significance in relation to other types of pulmonary complication.

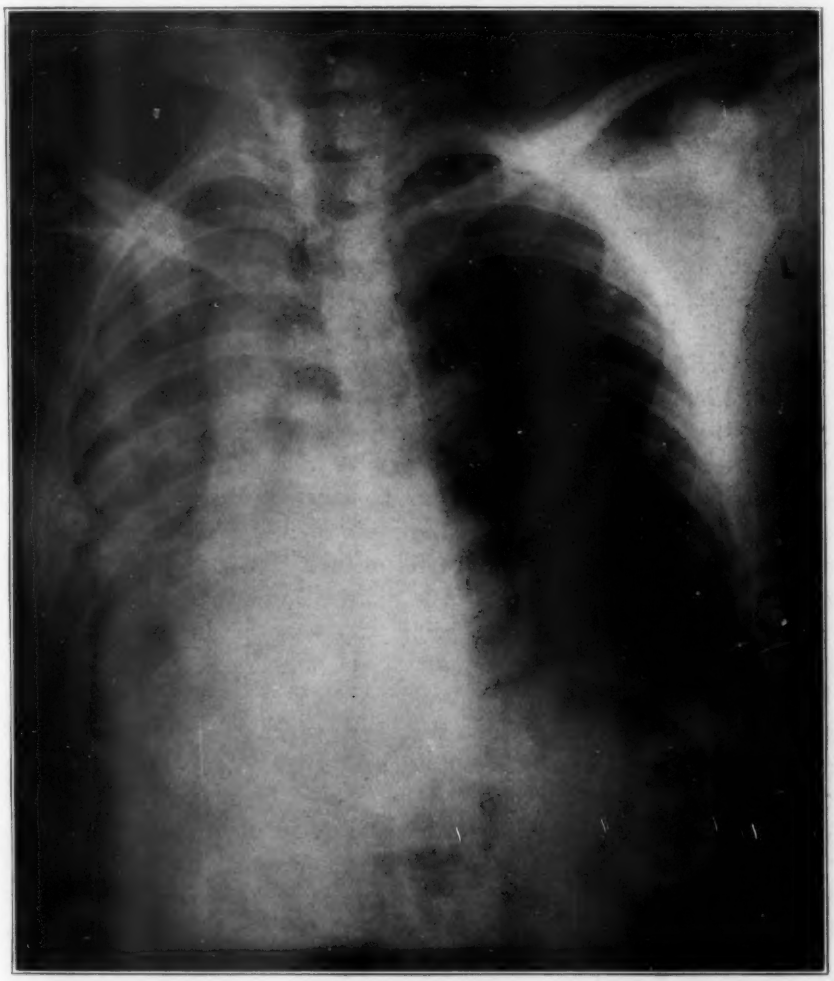

Fig. 2 (Case 38).—Roentgenogram taken sixteen hours after Figure 1; physical signs are obvious now, and the diagnosis can be made by inspection of the patient; contrast in the opacity of the right base with the clearness in the previous film.

This condition was originally described as a postoperative complication under the title of "Massive Collapse of the Lung."[6] The idea

6. Pasteur, W.: Massive Collapse of the Lung, Lancet **2**:1351, 1908.

conveyed by the term massive is essential in order to distinguish the condition, produced when at least the major part of one lobe is involved, from the lobular atelectasis that occurs in areas too small to present decisive symptoms and in which emphysematous patches are usually interspersed. Because of the fact that this deflation occurs in both massive and small areas, and since the term atelectasis is accepted in clinical as well as pathologic conditions,[7] it would seem wiser to substitute this term for that of collapse of the lung.

History.—In his Bradshaw lecture of 1908, W. Pasteur[6] reported finding at necropsy a partially airless condition of one or both lower lobes of the lung in three postoperative patients dying with peritonitis. He concluded his address with the suggestion, made for the first time, that some of the postoperative pulmonary complications consisted in a collapse of lung tissue. The first case of postoperative massive atelectasis to be recognized clinically was described by Pasteur in 1910,[8] and his articles of 1911 and 1914 record several typical examples of the condition.[9] Following 2,000 operations at the Middlesex Hospital in 1911-1912, sixty-five pulmonary complications were recognized, and, of these, sixteen were diagnosed as massive collapse of the lung.[10] Each one of the latter followed laparotomy, and usually there was no infection present. Only one other report of postoperative massive atelectasis appeared before the war. In 1914, Elliott and Dingley[11] reported seeing eleven cases in which this condition appeared after operation. Their examples also followed abdominal operations in which no other complication was present.

At an early period of the World War, it was noticed that chest injuries often gave rise to unusual signs which were inexplicable as due to the recognized sequelae of such injuries. A new consideration of such chest injuries developed from the following bizarre findings: (1) A high diaphragm on the affected side in cases of sterile hemothorax; (2) an immobile, but retracted chest wall with displacement of the heart toward the hemothorax; (3) dulness, with displacement of the heart toward the affected side in simple contour wounds without injury to the pleura or ribs; (4) the latter syndrome on the opposite, uninjured side of the chest, and (5) these intrathoracic signs after an injury that

7. Hamman, L. V.: Diseases of the Lung, Oxford Medicine **2**:65, 1920.

8. Pasteur, W.: Active Lobar Collapse of the Lung After Abdominal Operations, Lancet **2**:1080, 1910.

9. Pasteur, W.: Postoperative Lung Complications, Lancet **1**:1329, 1911; Massive Collapse of the Lung, Brit. J. Surg. **1**:587, 1914.

10. Pasteur (Footnote 9, second reference).

11. Elliott, T. R., and Dingley, L. A.: Massive Collapse of the Lungs Following Abdominal Operations, Lancet **1**:1305. 1914.

was entirely removed from the chest.[12] After repeated observation of these signs in war wounds, investigators concluded that massive atelectasis was the responsible factor. Bradford found this complication to occur in at least from 5 to 10 per cent. of all chest wounds when it was carefully looked for; and Barling[13] states that it is an almost constant phenomenon in penetrating wounds of the chest.

The knowledge thus gained from war wounds gradually became disseminated, and has resulted in a rediscovery of massive atelectasis as a postoperative complication. Scholtz,[14] in South Africa, called attention to the apparently identical syndrome following operation, which had become familiar after war injuries, and he reported the details in one example after herniotomy. Scrimger,[15] in 1921, reported the first cases of postoperative massive atelectasis to appear in the American literature. He found seven examples of this complication in a series of 540 consecutive operations. During the following year, he saw three more cases.[16] In the three years since 1921, twenty such cases have been reported as diagnosed in several different clinics.[17] Even so it is not generally recognized that postoperative massive atelectasis presents a striking and characteristic clinical picture, and is probably a common postoperative pulmonary complication. For example, Riesman[18] recently referred to it as a very rare postoperative condition. This impression is due to our failure to diagnose this complication, rather than to its actual rarity. When the condition is once recognized in a

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12. Bradford, J. R.: Massive Collapse of the Lung as a Result of Gunshot Wounds, with Especial Reference to Wounds of the Chest, Quart. J. Med. **12**:127, 1918-1919.

13. Barling, S. G., and Morrison, J. T.: Manual of War Surgery, New York, Oxford University Press, p. 170.

14. Scholtz, C. J.: Massive Collapse of the Lung, M. J. South Africa **16**:202 (June) 1921.

15. Scrimger, F. A. C.: Postoperative Massive Collapse of the Lung, Surg., Gynec. & Obst. **32**:486 (June) 1921.

16. Scrimger, F. A. C.: Postoperative Massive Collapse of the Lung, Am. J. Surg. (Anesthesia Supp.) **36**:53 (April) 1922.

17. Elwyn, H., and Girsdansky, J.: Postoperative Massive Collapse of the Lung, J. A. M. A. **79**:718 (Aug. 26) 1922. Hirschboeck, F. J.: Postoperative Massive Collapse of the Lung, Am. J. M. Sc. **164**:268 (Aug.) 1922. Gwyn, N. B.: The Clinical Side of the Condition Spoken of as Massive Collapse of the Lung, Tr. A. Am. Phys. **38**:411, 1923. Leopold, S. S.: Postoperative Massive Pulmonary Collapse and Drowned Lung, Am. J. Med. Sc. **167**:421 (March) 1924. Ritvo, M.: Massive Collapse of the Lungs, Am. J. Roentgenol. **11**:337 (April) 1924. Elwyn (Footnote 4). Lee (Footnote 5).

18. Riesman, D.: Diagnostic Problems in Pneumonia, J. A. M. A. **82**:1256 (April 19) 1924.

given clinic, successive cases have soon been discovered there. Thus, at the Middlesex Hospital, no examples of massive atelectasis were diagnosed clinically before 1908 when Pasteur first discovered it as a postoperative complication, while at least fifteen cases were recognized in the succeeding two and a half years. And Scrimger found seven instances of it in one year in a single clinic.

II. POSTOPERATIVE MASSIVE ATELECTASIS—A CLINICAL ENTITY

In order to have a comprehensive view of this condition, all the reported cases have been studied. From an analysis of these, it is possi-

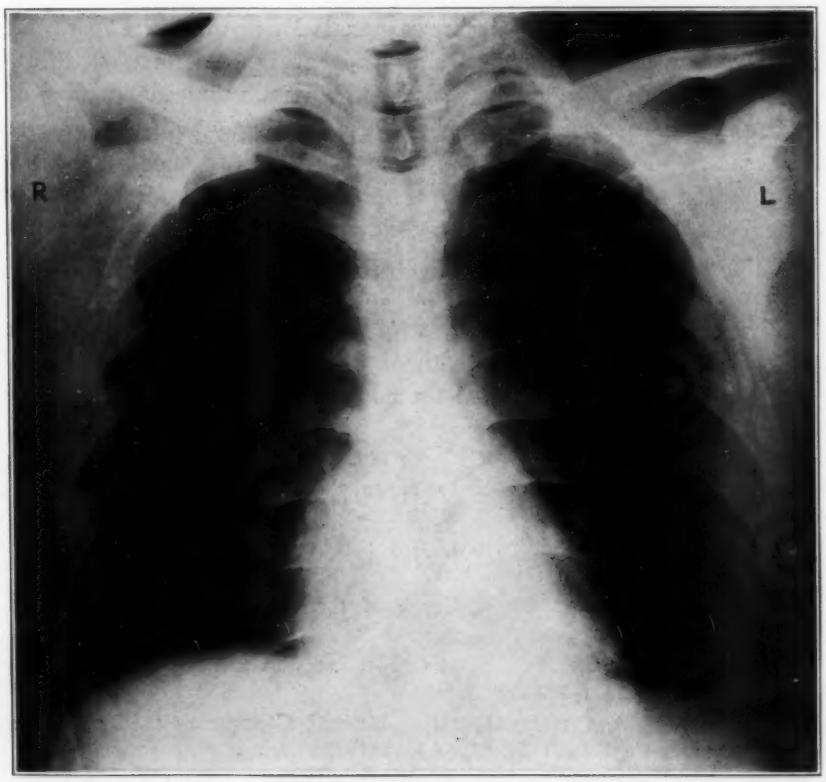

Fig. 3 (Case 39).—Postoperative massive atelectasis, right, latent type half an hour before operation for appendicitis with abscess.

ble to determine certain fundamental characteristics and the limits of variability of the syndrome. Sixty-four instances have been recorded in the literature; only thirty-eight of these, however, are reported in sufficient detail to be of value for analytic purposes. Four additional cases which I have recently had the opportunity to study are added. The protocols of these four cases are contained in this paper.

The reported cases of massive atelectasis following war wounds have been purposely excluded from this study for two reasons: (1) Our uncertainty as to the identical nature of the mechanism of the condition following trauma and operation, and (2) the difficulty of prolonged observation under the war conditions. Certain facts demonstrated by these posttraumatic cases and having a direct bearing on the consideration of the postoperative complication are cited in later sections. Our

Fig. 4 (Case 39).—Condition three days after operation; the patient had complained of no respiratory symptoms; thoracic examination was made on account of a few scattered râles present before operation. Comparison with Figure 3 should be made. These roentgenograms are reproduced through the courtesy of Dr. P. F. Butler, roentgenologist of the Boston City Hospital.

conclusions then are drawn from an analysis of forty two cases of postoperative massive atelectasis reported from twelve different sources. Tables 1 and 2 give a résumé of the significant data, and reference to individual cases will be made by their numbers in these tables.

On the basis of the observations made by so many independent investigators, we are able to form a satisfactory conception of this complication; its symptoms, clinical course, and the extreme manifestations that it presents. All the reports are in unanimous agreement about certain fundamental symptoms and signs which have been invariably present. These constitute a definite clinical entity. From

TABLE 1.—*Preoperative Condition* *

Case	Author and Year	Age	Sex	Month	General Condition	Condition of Lungs	Maximum Temp.
1	Pasteur, 1910, 1911, 1914.....	23	♂	Nov.	Healthy	.....	.....
2	Pasteur, 1910.....	37	♂	Jan.	Infectious wound	.....	.....
3	Pasteur, 1910, 1914.....	24	♀	April	Strong	.....	.....
4	Pasteur, 1910, 1914.....	45	♀	June	Good physique	.....	.....
5	Pasteur, 1910.....	49	♀	June	Dyspnea	.....	.....
6	Pasteur, 1911, 1914.....	..	♀	Feb.	Dyspnea	Rhonchi	.....
7	Pasteur, 1911.....	18	♀	Nov.	Acute abdomen	.....	.....
8	Pasteur, 1911, 1914.....	12	♂	.....	Healthy	Weak breath sounds at right base	.....
9	Pasteur, 1914.....	23	♀	.....	Good physique	Weak breath sounds at left base	.....
10	Elliott, Dingley, 1914.....	31	♂	July	No cough	.....	.....
11	Elliott, Dingley, 1914.....	23	♂	.....	Acute abdomen	Weak breath sounds at right base	100
12	Scholtz, 1921.....	37	♂	Jan.	.....	.....	.....
13	Scrimger, 1921.....	..	♂	July	.....	.....	.....
14	Scrimger, 1921.....	..	♂	Feb.	Good	Negative	.....
15	Scrimger, 1921.....	..	♂	Nov.	Chilled	.....	.....
16	Scrimger, 1921.....	..	♂	Jan.	.....	.....	.....
17	Scrimger, 1921.....	..	♂	Apr.	Cough; expectoration	Negative	.....
18	Scrimger, 1921.....	..	♂	June	.....	.....	.....
19	Scrimger, 1921.....	..	♂	Sept.	.....	.....	.....
20	Scrimger, 1922.....	30	..	Nov.	.....	.....	.....
21	Scrimger, 1922.....	21	..	Apr.	Ill	.....	.....
22	Scrimger, 1922.....	13	..	Mar.	Suffocation attacks	.....	.....
23	Elwyn; Giradansky, 1922...	14	♂	Apr.	Stab wound	Negative	.....
24	Hirschboeck, 1922.....	38	♀	Nov.	Good	.....	98.6
25	Hirschboeck, 1922.....	49	♂	June	.....	.....	100
26	Gwyn, 1923.....	20	♂	Nov.	.....	.....	.....
27	Gwyn, 1923.....	..	..	.....	.....	.....	.....
28	Gwyn, 1923.....	26	♂	.....	Acute abdomen	Wheezes	.....
29	Elwyn, 1924.....	32	♂	Feb.	.....	Negative roentgen-ray examination	.....
30	Elwyn, 1924.....	35	♂	May	.....	.....	.....
31	Leopold, 1924.....	29	..	Feb.	.....	.....	99.2
32	Leopold, 1924.....	22	..	Mar.	.....	.....	97
33	Leopold, 1924.....	25	..	Mar.	.....	.....	97.8
34	Leopold. 1924.....	24	..	Feb.	.....	.....	98.2
35	Lee, 1924.....	16	♂	Mar.	.....	.....	.....
36	Ritvo, 1924.....	28	♂	.....	Healthy	Negative	98.6
37	Scott, 1924.....	18	♂	Dec.	Healthy	Negative	99
38	Scott, 1924.....	20	♂	Jan.	Healthy; cough	Negative	98.8
39	Scott, 1924.....	19	♂	Jan.	Robust	Râles	99.8
40	Scott, 1924.....	26	♂	April	Vigorous; urticaria	Negative	100.2

* In this table, ♂ signifies male; ♀, female.

them, we may define postoperative massive atelectasis as a febrile complication arising within a few days of operation, the characteristic and unique feature of which is the combination of the signs of a unilateral pulmonary consolidation with displacement of the heart to the affected side.

Onset and Symptoms.—The onset of symptoms occurred within twenty-four hours of operation in half the cases and after forty-eight

TABLE 2.—*Postoperative Massive Atelectasis* *

Case	Operation	Anesthetic†	Side	Onset in Days	Symptoms: Cyanosis	Dyspnea	Cough	Expectoration	Pain, Chest	Pain, Wound	Maximum: Temperature	Pulse	Respiration	White Blood Cells	Asymmetry of Chest	Limitation of Respiration Movement	Dulness	Suppressed Breath Sounds	Bronchial Breath Sounds	Râles	Hyperresonance on Opposite Side	Displacement of Heart to Affected Side	Roentgen Ray	Duration of Fever	Duration of Signs
1	Left inguinal hernia	G.E.C.	R	1	+	+	—	—	+	—	103	132	—	—	—	+	+	+	—	0	+	+	—	1	17
2	Right inguinal abscess	—	R	4	—	+	—	+	+	—	101	—	—	—	+	—	+	+	+	Both sides	+	+	—	1	11
3	Salpingectomy	—	L	3	+	+	+	+	+	+	104.8	132	45	—	—	—	+	+	—	Crepitant	+	+	—	3	5
4	Cholecystectomy	G.E.C.	R	3	+	+	+	+	0	+	102.6	140	40	—	—	+	+	+	+	0	+	+	—	4	10
5	Cholecystectomy	—	R	2	+	+	+	+	—	+	101	120	44	—	+	—	+	+	+	Rhonchi, crepitant	+	+	—	9	9
6	Hysterectomy	Chlor.	R	2	—	+	—	+	+	—	102.6	120	32	—	+	+	+	+	+	Rhonchi, both sides	—	+	—	5	10
7	Peritonitis	Chlor.	R	3	0	0	+	+	0	—	101	104	24	—	—	—	+	+	+	Medium, fine on right	—	+	—	—	—
8	Appendectomy	G.E.C.	R	2	0	0	+	0	0	0	100	—	—	—	—	+	+	+	+	0	—	+	—	1	3
9	Appendectomy	—	L	2	0	0	0	0	0	0	+	130	30	—	+	+	+	—	+	0	—	+	—	2	5
10	Bilateral inguinal hernia	Chlor.	R	6	+	+	—	+	+	—	101	144	64	—	—	+	+	+	+	—	—	+	+	8	8
11	Appendectomy; drain	Spinal	R	2	0	—	—	+	+	—	102	116	40	—	—	—	+	+	—	Moist, both sides	—	+	0	—	4
12	Left inguinal hernia	G.E.	R	1	—	+	+	+	+	—	+	+	+	—	+	+	+	+	+	Few	—	+	+	—	—
13	Left inguinal hernia	—	R	1	—	+	+	+	+	—	—	—	—	—	—	+	+	+	—	—	—	+	—	—	—
14	Appendectomy	—	L	3	—	+	+	+	—	—	102.2	120	20	—	—	—	+	+	—	—	—	+	+	1	—
15	Strangulated hernia	—	L	1	—	+	+	—	+	—	101	120	36	—	+	+	+	+	—	—	—	+	+	—	2
16	Right inguinal hernia	—	R	—	—	+	+	—	—	—	101	108	28	—	+	—	+	—	+	Wheezes, right	—	+	+	4	4
17	Right inguinal hernia	—	R	2	—	0	—	—	—	—	100	112	36	—	—	—	+	+	—	—	—	+	+	—	—
18	Appendectomy	—	R	2	—	+	+	—	+	—	101	120	40	—	—	+	+	+	—	—	—	+	+	—	—
19	Hemorrhoidectomy	—	L	1	—	0	+	+	+	—	100	—	28	—	—	+	+	+	—	—	—	+	+	—	—
20	Appendectomy; drain	G.E.	R	1	—	+	+	+	—	—	102	120	30	—	—	+	+	+	—	—	—	+	+	—	—
21	Appendectomy; drain	G.E.	R	1	—	+	+	+	—	—	100	160	30	—	—	+	+	+	—	—	—	+	+	—	—
22	Appendectomy; drain	G.O.	L	1	+	+	+	+	+	—	104	140	40	—	—	+	+	—	—	—	—	+	+	—	4
23	Laparotomy	—	R	1	+	+	+	+	—	—	103	—	—	—	+	+	+	+	+	Few	+	+	+	5	13
24	Appendectomy; oophorectomy	—	L	2	—	—	—	—	—	—	101	130	—	21,200	—	+	+	—	+	Late, mucous	—	+	—	—	—
25	Appendectomy; drain	—	R	1	—	+	—	—	—	—	104	120	—	—	—	—	+	—	+	Few, late	—	+	—	2	—
26	Appendectomy	Ether	R	1	—	+	+	+	+	—	+	+	—	—	—	+	+	+	+	Late	—	+	+	—	14
27	Appendectomy	Ether	L	1	+	+	+	+	—	—	+	+	—	—	+	+	+	+	+	Late	—	+	0	—	—
28	Appendectomy; drain	—	R	1	+	+	+	—	—	—	—	—	—	—	+	+	+	+	+	Crepitations	—	+	+	—	10
29	Left inguinal hernia	G.E.	R	1	—	—	+	—	+	—	—	—	—	—	—	—	+	+	—	Late	—	+	+	—	—
30	Colectomy	G.O.	R	1	—	—	—	—	—	—	102	—	—	—	—	—	+	+	—	—	—	+	+	—	—
31	Ventral hernia	Ether	R	1	+	+	—	—	—	—	102.8	146	44	34,000	—	+	—	+	—	—	—	+	+	4	5
32	Left inguinal hernia	Ether	R	3	+	+	—	—	—	—	102	120	40	—	—	+	—	+	—	—	—	+	+	6	—
33	Right inguinal hernia	Ether	R	2	+	+	—	—	—	—	104.6	120	40	—	—	+	—	+	—	—	—	+	+	7	7
34	Left inguinal hernia	Ether	L	1	+	+	—	—	—	—	103	114	36	—	—	+	—	+	—	—	—	+	+	2	—
35	Appendectomy; drain	—	R	3	—	—	—	+	+	0	—	—	—	—	—	+	+	+	—	Course on right	+	+	—	—	14+
36	Ventral hernia	G.E.	R	2	—	+	+	+	+	—	102	105	30	11,400	—	+	+	—	+	Late, fine	+	+	+	4	9
37	Appendectomy	Ether	R	2	+	+	+	+	+	0	102.8	134	40	23,000	+	+	+	+	—	Late, sonorous	+	+	+	6	9
38	Appendiceal ventral hernia	Ether	R	2	+	+	+	+	+	+	103.6	130	52	14,200	+	+	+	+	+	Intermittent	+	+	+	4	9+
39	Appendectomy; drain	G.E.	R	2	0	0	+	+	0	0	102	120	30	—	+	+	+	+	+	Intermittent	+	+	+	—	9
40	Appendectomy	Ether	R	1	+	+	+	+	+	0	102	106	34	18,000	+	+	+	+	+	Late on right	+	+	+	6	15+

* In this table, + signifies present; 0, absent, and —, that data are not given. All the patients recovered.
† In this table, G.E.C., signifies gas-ether-chloroform; Chlor., chloroform; G.E., gas-ether; G.O., gas-oxygen; R, right; L, left.

hours in only 20 per cent. of the cases. In fact, the patient may show some symptoms as soon as conscious after the anesthetic, as, for example, Case 26 of Gwyn.[19] Ten per cent. are classified as latent, as they complain of practically no symptoms. In all the other cases the involvement of the respiratory system was suggested by the presence of more than one of the following symptoms: pain in the chest, cough, expectoration, dyspnea, tachypnea and cyanosis. In several instances, a sudden, severe attack of respiratory distress so acute as strongly to suggest a pulmonary embolus called attention to the complication. Usually, in these cases, premonitory symptoms had been present, but had received scant attention. Very commonly, the feature to attract notice is a sudden, sharp rise in the temperature and the pulse and respiratory rates, usually accompanied by a painful, hacking cough and some dyspnea. And in all cases, even in the latent ones, there is fever and tachycardia. Only 12 per cent. had a maximum temperature below 101, none below 100. The highest temperature was 104.8. The maximum pulse rate varied between 108 and 160. The respiratory rate was 30 or above in all but three cases.

The brief period during which some of these manifestations are present in the milder cases is illustrated in Case 11 of Elliott and Dingley.[11] Here, the pulse and respiratory rates remained elevated for only four hours. Usually, however, the temperature, pulse and respiration charts show elevations lasting for several days.

Dyspnea, cough and pain in the chest are all common symptoms; but they occur to varying degrees, and any one of them may be absent in an outspoken case. Expectoration is frequently a later symptom, often appearing a day or two after the others. It may be present at the onset, or it may be absent altogether. In some instances, it is definitely blood stained, as in Cases 14 and 20 of Scrimger.

Physical Signs.—It will be noted that these symptoms are not pathognomic, but are common to several other postoperative pulmonary complications. Massive atelectasis is differentiated from all the latter by its physical signs. It is agreed by all observers that there is unilateral dulness with displacement of the heart to the affected side. No other acute condition is known to present this combination. The area of dulness varies in extent. It may involve the whole of one lung, though usually some resonance is found in the supraclavicular fossa and often over much of the anterior aspect. The dulness is most marked usually at the base posteriorly. The extent to which the heart is displaced varies greatly. In all the cases tabulated, it was striking. With the collapse on the right side, the heart may be entirely to the right of the midline.

19. Gwyn (Footnote 17, third reference).

When the left lung is affected, the apex beat has been found even in the axilla. Obviously, these are the extremes; in any well marked instance, though, the displacement toward the affected side is 3 or 4 cm.

Over the collapsed area, the breath sounds are altered in one of two directions; either they are (1) greatly diminished or completely suppressed, or (2) bronchial in character. And, frequently, the same case will show both modifications at different times or in different parts of the chest. Probably, the majority of cases have diminished breath sounds at first, followed by bronchial breathing. Even when the breathing is largely suppressed there is often a bronchial quality to the sound that is heard. Apparently though, in some cases, loud bronchial breathing may be present continuously from the onset, or may be followed by suppressed breath sounds. The bronchial breathing may be tremendously loud and is usually accompanied by bronchophony and pectoriloquy, while the suppressed breath sounds are associated with diminished vocal fremitus. The exact nature of the signs at the time of the onset is rather difficult to determine from most of the cases recorded in the literature, as the condition was not recognized until after it had become well advanced. Three recent cases that were observed while the condition was developing showed marked diminution in breath sounds at first; in one of them, this continued throughout, while, in the other two, the suppressed sounds were replaced by loud bronchial breathing on the next and on the third day, respectively, after the onset.

Râles are frequently altogether absent over the dull area in the early stages, even when loud bronchial breathing is heard. In the majority of cases, they occur at a later period, and are apt to be prominent when the atelectasis is beginning to subside. They are usually not of the fine subcrepitant variety, so frequent in early lobar pneumonia, but are the musical or coarse râles that we associate with bronchitis and asthma.

There are certain other signs which are due to the collapsed condition of the underlying lung, and are of considerable assistance in picking out the cases of massive atelectasis. If the patient is placed flat on his back, a marked asymmetry of the two sides of the thorax will be noted. The affected side is strikingly depressed (Fig. 5 and section on Differential Diagnosis), and its respiratory excursion is curtailed; in fact, when the complication is well developed there is usually little respiratory movement noticeable on the affected side.

In spite of the limited movement of the chest wall on the affected side, the lateral costal margin moves outward farther on this side than on the opposite one, as called to our attention by Dr. C. F. Hoover. This phenomenon is probably dependent on the fact that the diaphragm is elevated on the collapsed side. This high position of the diaphragm

may be demonstrated when the collapse is on the left side by percussion of Traube's space and by the absence of liver dulness in the epigastrium when the atelectoris is on the right side.

On the unaffected side of the chest there is a drumlike, hyperresonant note on percussion, and the breath sounds are usually exaggerated but of normal type. When the atelectasis is on the right, the superficial cardiac dulness is obliterated. In our own cases, we have found that there are usually sonorous râles on the unaffected side at some stage of the condition, though this fact is not brought out in the literature.

In addition to these local signs, most of the patients show a marked degree of cyanosis, and in one instance (Case 12 of Scholtz) there was an icteric tint to the skin and conjunctivae on the day after the onset.

Location of the Atelectasis.—In three fourths of the postoperative cases, the massive collapse has occurred in the right lung. The area involved may include the whole lung, or it may involve only part of it. Usually, the extreme apex of the lung is relatively clear and the base is involved, though there are definite cases apparently limited chiefly to the upper lobe (Fig. 10).

Roentgen-Ray Findings.—The roentgen-ray examination of the chest is of great assistance in establishing the diagnosis when the physical signs are not yet clear. This is of particular importance early in the development of the complication, and it has also been shown that there are all gradations in the extent of the collapse. Probably there are minor degrees in which the cardiac displacement can be determined only by roentgen ray. The examination of postoperative patients is carried out under conditions unfavorable to the demonstration of finer changes in the thoracic physical signs. The patient cannot sit up for the determination of the cardiac position. To that inherent difficulty is added the obliteration of the superficial cardiac dulness by a distended left lung when, as is usually the case, the collapse is on the right side. Consequently, it becomes impossible to be dogmatic about a slight change in the cardiac position. Thus, for example, in Case 38, on the day after operation when the symptoms appeared, the diagnosis of massive atelectasis could not be definitely established until the roentgen-ray plate verified the displacement of the heart (Fig. 1). On the following morning, the complication could be diagnosed merely by inspection of the patient. Furthermore, information of great importance in regard to the mechanism of the collapse is obtained by the evidence of serial roentgen rays beginning early in the development of the condition. We cannot here dwell on the details of the roentgenologic aspects of postoperative massive atelectasis. Suffice it for our purposes to state that the area of dulness shows in the roentgen-ray film as an area of increased density which is sometimes so extreme that the inter-

Fig. 5 (Case 38).—Postoperative Massive atelectasis, right, obvious depression of affected side of the chest after the patient is placed flat on his back; contrast is even more striking on respiration, the right thorax moving but little, while the left has a large excursion. Comparison with Figure 3 should be made.

Fig. 6 (Case 38).—Position these patients nearly always assume for comfort; in it, the asymmetry and respiratory restrictions are no longer obvious. Comparison with Figure 5 should be made.

spaces cannot even be distinguished, and that the displacement of the heart and mediastinum (shown by the trachea) graphically records the increased negative pressure on the collapsed side. The diaphragm on the affected side, when it can be seen, is high and, fluoroscopically, its respiratory excursion has stopped or is greatly limited.

Roentgen-ray examination demonstrates the clearing of the area of density, occurring at times so rapidly that consolidation or fluid could not have been the cause of the shadow. A remarkable illustration of this is found in a recent case, reported by Leopold,[20] in which the chest plates show on successive days an alternation of density and clearing, the position of the heart varying in a corresponding direction.

Clinical Course.—There are three fairly distinct forms of this condition dependent on the symptomatology: (1) fulminating; (2) moderate to severe, and (3) latent.

In the fulminating form, in several of the cases recorded, the respiratory distress came on in such an abrupt and severe manner that the examiner thought the result would be fatal within a few hours. Thus, in Case 28 of Gwyn,[19] the patient was reported as dying when the condition was first noticed. The complication, in such instances, has been interpreted at first as a pulmonary embolism on account of its fulminating character. This is referred to in the literature as a "collapse attack." As a matter of fact, the condition had probably been developing for some time before the sudden respiratory distress occurred, as respiratory symptoms had preceded the latter. The alarming symptoms of the attack, however, are greatly ameliorated within twenty-four hours, and the further course of this type is identical with the next one.

In the majority of cases in the moderate to severe form, symptoms pointing to the respiratory system, namely, pain in the chest, cough or dyspnea appear and often become progressively more troublesome, accompanied by a sharp rise in the temperature, pulse and respiratory rates. The clinical chart suggests the onset of a postoperative pneumonia. The physical sign, at this time, may not be absolutely conclusive, but a chest plate will demonstrate the cardiac displacement, and the condition will develop obvious signs within twenty-four hours. Contrary to the commonly accepted view in the literature, I believe on the basis of our observations that, in the early stages, the degree of pulmonary collapse progresses for several hours, and that this fact will be proved if roentgen-ray plates are taken when respiratory symptoms and the temperature elevation are first noted, and then taken again after twelve and twenty-four hours (Figs. 1 and 2).

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20. Leopold (Footnote 17, fourth reference).

Respiratory symptoms usually do not last more than a few days. The elevation of temperature lasts from a few hours to eleven days, and the physical signs usually begin to clear up with the symptomatic improvement. The complete disappearance of all intrathoracic signs often takes two weeks or more. No case of typical unilateral collapse of the lung, reported in detail to date, has been fatal unless compli-

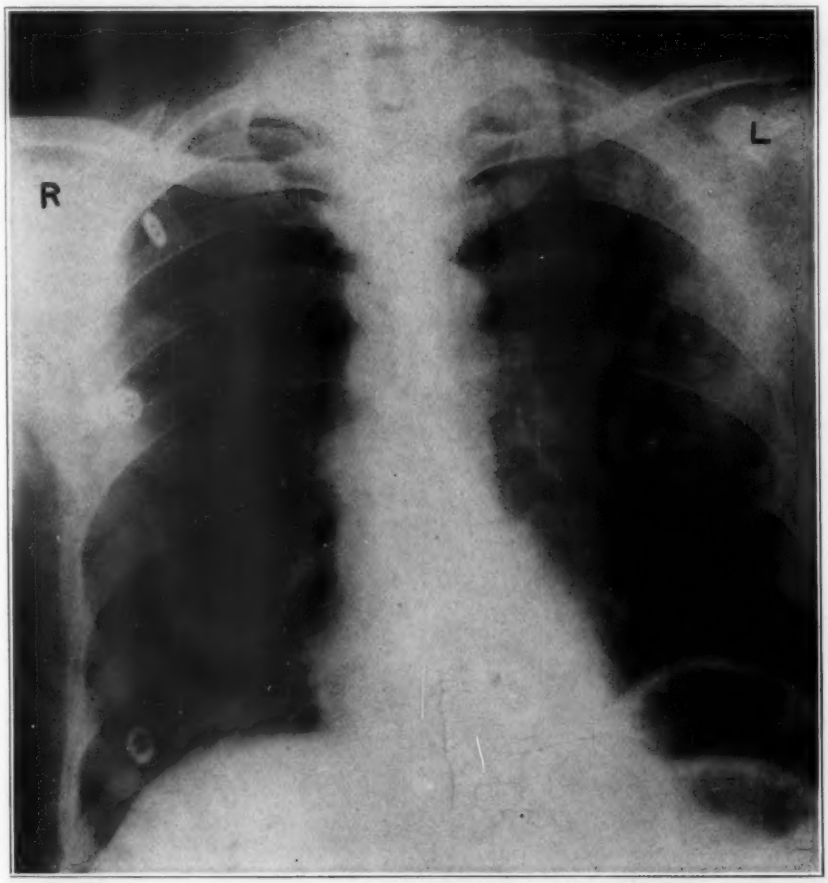

Fig. 7 (J. L.).—Postoperative massive atelectasis, right, complicated by bronchopneumonia which was fatal, three days before posterior gastrojejunostomy for duodenal ulcer and pyloric stenosis; lung fields clear except for small calcified areas in first left interspace (probably old tuberculous lesions).

cated by pneumonia (sections on complications). This is true in spite of the alarming nature of the "collapse attack." An interesting course has been observed by Leopold in one case of postoperative massive atelectasis. There was alternate collapse and reexpansion on successive days over a period of six days. This was verified by roentgen ray.

After a variable period not extending beyond from three to four weeks, the atelectasis has completely disappeared without residual symptoms or signs.

The condition terminates in one of three ways, by: (1) lysis, (2) complication or (3) crisis.

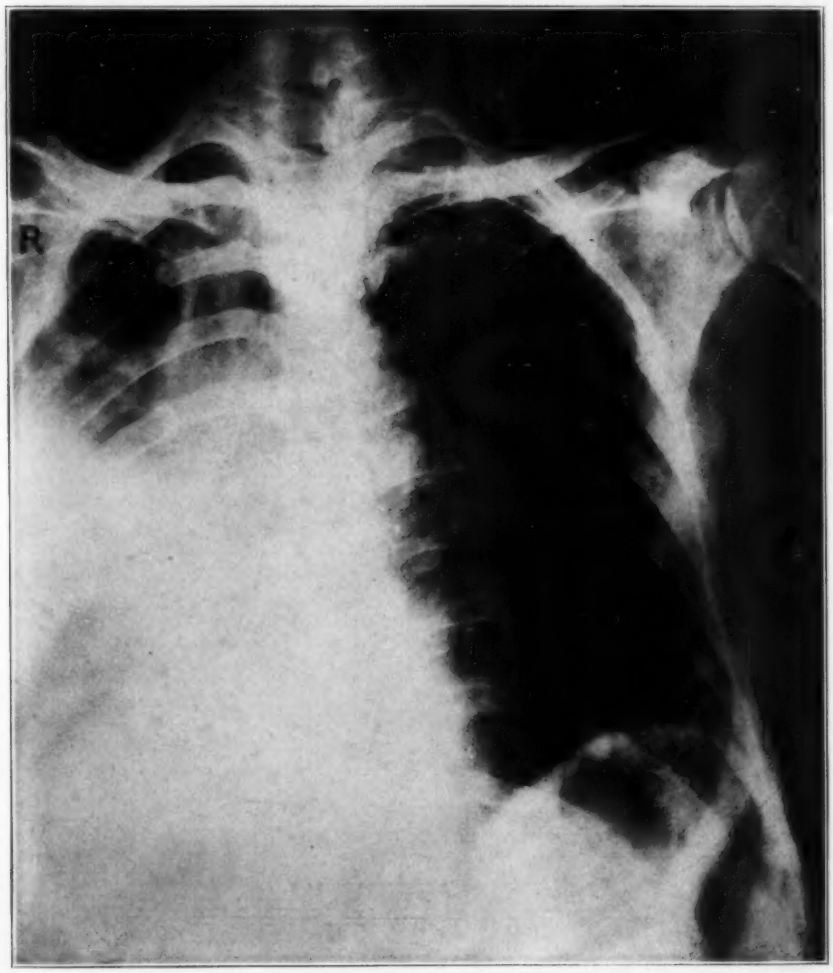

Fig. 8 (J. L.)—Ten days after operation, the right upper part of the lung relatively clear; the heart and mediastinum are well displaced to the right. Comparison with Figures 7 and 9 should be made.

1. By lysis. The usual course is for the symptoms gradually to abate, after being present for a few days, accompanied by a change in the signs. Frequently, at this time, râles become a prominent feature which may be marked on both sides of the chest. The entire disappear-

ance of signs takes another week or more, with fluctuations in the character of the breath sounds and the râles from day to day.

2. By complication. The complications of massive atelectasis will be discussed in a later section. When such arise, the further course then becomes largely that of the complication. All the fatal cases so far reported seem to come in this group of cases having a bronchopneumonia superimposed on the massive atelectasis.

3. By crisis. In a number of cases, this condition has been terminated in an abrupt manner by crisis. Symptoms and most of the signs have cleared up suddenly during the course of a few hours. This, in some instances, has apparently followed the coughing up of a mass of tenacious, mucopurulent secretion.[15]

An interesting, even if somewhat select, group of cases is formed by those in which this complication has occurred practically without respiratory symptoms, and which would undoubtedly have been entirely overlooked except for the accidental discovery of physical signs in the chest. These patients have some fever and tachycardia. There are two cases reported that clearly fall within this group; and four others about which the data is insufficient to make certain whether they should be included. Patient 8, reported by Pasteur, had no symptoms aside from an occasional short cough; his temperature was 100 F. the day after operation, but was normal the following day when signs of collapse were discovered on routine examination of the chest. Patient 39 had complained of no symptoms, but his chest was examined on account of the preoperative discovery of a few râles. He had all the typical signs of massive atelectasis, and the diagnosis was confirmed by roentgen ray (Figs. 3 and 4). By having this patient lie on the unaffected side (left), marked respiratory distress was brought out within two minutes. The dyspnea forced him to return to the dorsal position, and it then again disappeared. In these latent cases, the physical signs clear up within a few days leaving no residuum. If care is taken to examine all postoperative patients that develop any fever, more latent cases will assuredly be found.

Predisposing Factors.—1. Age. This condition is reported in patients from 12 to 49 years of age. It will probably be found to occur also in children when it is looked for. Forty per cent. of the cases so far recorded have occurred in the third decade.

2. Sex. A marked majority of the cases analyzed were in males. No importance at all can be attached to this preponderance as many of the cases reported [21] were drawn from a military population. Pasteur

21. Scrimger (Footnote 15). Gwyn (Footnote 17, third reference). Leopold (Footnote 17, fourth reference).

records more cases in female than in male patients. In this connection, the only certain deduction that can be drawn is that massive atelectasis occurs after operation in both sexes.

3. Season. This complication occurs in all seasons of the year. There is not the very striking seasonal incidence that attaches, for example, to lobar pneumonia. As many cases are recorded in June and July as in December and January. No further deductions in this connection are justified on the basis of the meager data available at present.

4. General preoperative condition. Most of the patients have been vigorous individuals; a number have had an acute appendicitis; none of them have been cachectic or the subject of a very chronic infection, and, surely, a preoperative febrile condition is not usual.

5. Previous lung disease. Apparently, in most of the cases, there had been no abnormality observed by the routine physical examination of the chest. This has been checked in some cases by roentgen-ray examination of the chest, so it seems safe to conclude that a preexistent pulmonary pathologic condition is not a necessary factor in causing postoperative massive atelectasis, and probably not even a usual one. A few of the patients have had râles in the chest, and at times weak breath sounds have been noted before operation over the base of the lung that collapsed after operation.

Differential Diagnosis.—Pasteur diagnosed over fifteen cases of massive atelectasis in the two and a half years following his discovery of the condition; previous to that time, none had been recognized. The question naturally arises: With what conditions had this been confused? Most of the unrecognized cases are called pneumonia, as the pulmonary signs may exactly resemble a lobar pneumonia, or confluent bronchopneumonia, except for the usual absence of subcrepitant râles. When this diagnosis is made, the displacement of the heart is overlooked. This mistake, for example, was made by the house officer in our Cases 37 and 38.

With this point in mind, twenty cases classified as postoperative pneumonia in the Peter Bent Brigham Hospital, during the years 1922 and 1923, were reviewed. These all had at least one chest plate. Three of them were unmistakably examples of massive atelectasis. Two of these cases are cited in the section on Complications on account of points of unusual interest, the third was a typical, uncomplicated case. In these instances, we were fortunately in possession of control roentgen-ray films taken either a few days before or after the pulmonary complication, proving conclusively that the displacement of the heart toward the affected side was acute and not due to chronic fibrosis. In five of the others, the appearance strongly suggested the same condition, but owing to the lack of the control normal pictures and the lesser degree of cardiac displacement, this diagnosis could not be posi-

tively made. In view of the fact that the roentgen-ray examination was made often several days after the onset of the complication, apparently with the idea of discovering whether the condition was clearing up, some at least of the latter cases probably had had typical massive atelectasis. In every instance supposed to be postoperative pneumonia, the position of the heart must be accurately determined to avoid making

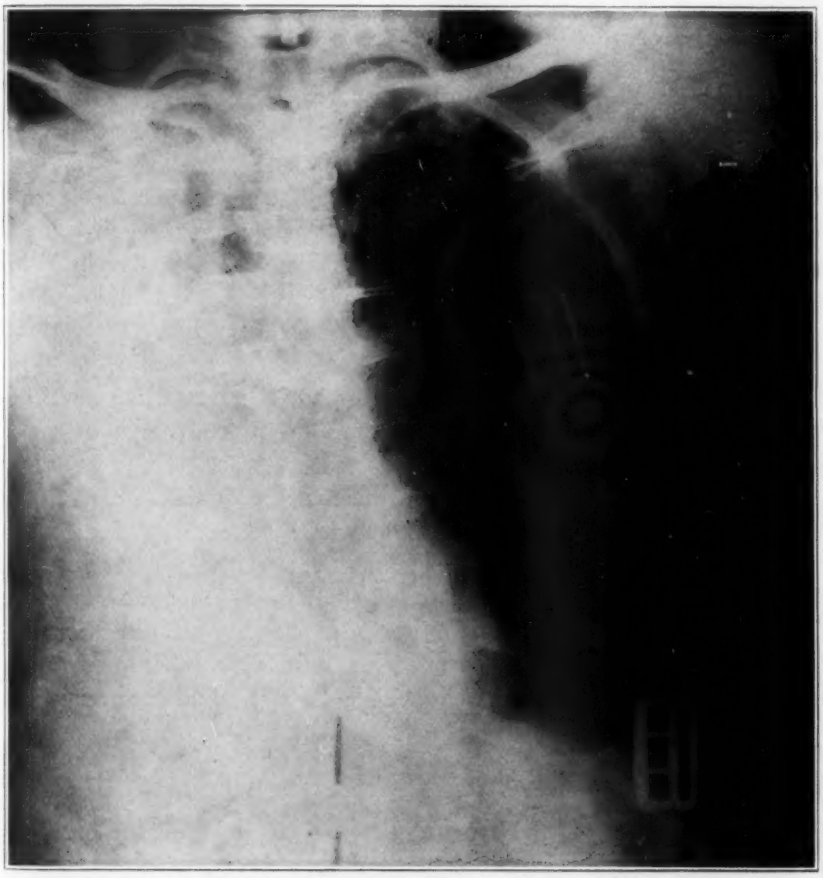

Fig. 9 (J. L.).—Thirteen days after operation, displacement of the mediastinum unchanged; opacity of the right apex in contrast to Figure 8 demonstrates the extension of the process; the patient had developed toxicity, and he died two days after this picture was taken. Presumably this is bronchopneumonia superimposed on massive atelectasis.

this most frequent mistake in the differential diagnosis of massive atelectasis.

When the collapse is on the right side, the heart may be entirely on the right side, and the condition has been mistaken for dextra cardia

on several occasions. The marked hyperresonance and obliteration of the cardiac dulness on the unaffected side has been mistaken for an acute pneumothorax, as in Case 31, reported by Leopold. The basal dulness has suggested a pleural effusion, and tapping has been done in several of these cases with negative results, on the basis of this diagnosis. Other mistaken diagnoses made for this condition are acute heart failure, pulmonary embolus and acute lung abscess. The first two of these have been suggested by the severe respiratory distress combined with cyanosis. The cardiac displacement should establish the differential diagnosis in these instances.

If the symmetry and respiratory movements of the chest are observed accurately with the patient lying exactly flat on his back, well marked cases of massive atelectasis can be detected easily, and suspected cases determined for roentgen-ray examination. In the former, depression of the chest (especially in the infraclavicular fossa) and limitation of the respiratory excursion on the affected side contrast sharply with the prominence of the opposite side and its great excursion with respiration. And all cases in which there is a respiratory lag on one side should be carefully studied and followed for the possible development of atelectasis. These signs are of great assistance when the position of the heart is difficult to map out. The marked asymmetry of the chest would surely attract attention to these cases were it not for a peculiarity of the condition. The patients are prone to lie on the affected side with a bending of the thoracic spine, and a tilting of the head in this direction. In such a position, the asymmetry and limitation of respiratory movement on the affected side are no longer conspicuous. These points are well illustrated in Case 38. Figure 5 shows the depressed right chest and prominent left side two days after operation when the massive atelectasis was well developed, while Figure 6 was taken at the same time, when the patient was in the natural position that he assumed for comfort.

One other prominent feature is of definite value in the discovery of these cases. This is the cyanosis that is present from the onset of acute symptoms. For example, on the first day after operation in Case 40 when the intrathoracic signs had not yet developed nearly to their maximum, the nurse had noted and asked why the patient was so blue. The cyanosis usually remains a number of days; its occurrence after operation should suggest the possibility of massive atelectasis to the observer.

In a manner similar to the mistaken diagnoses after operation, an associated massive atelectasis accompanying inflammatory lesions, both intrathoracic and intra-abdominal, is probably being overlooked. Pasteur saw "lobar collapse of the lung bases" in pleurisy, apical pneumonia

and hydrochloric acid poisoning, and Tidy [22] reported a case that occurred spontaneously, presumably associated with some pulmonary lesion masked by the collapse. The retraction of the chest wall, narrowing of the interspaces, and limitation of the respiratory movements on the affected side occurring early in the development of an acute empyema are quite possibly manifestations of a similar active collapse (as distinguished from the passive collapse of the lung from compression by

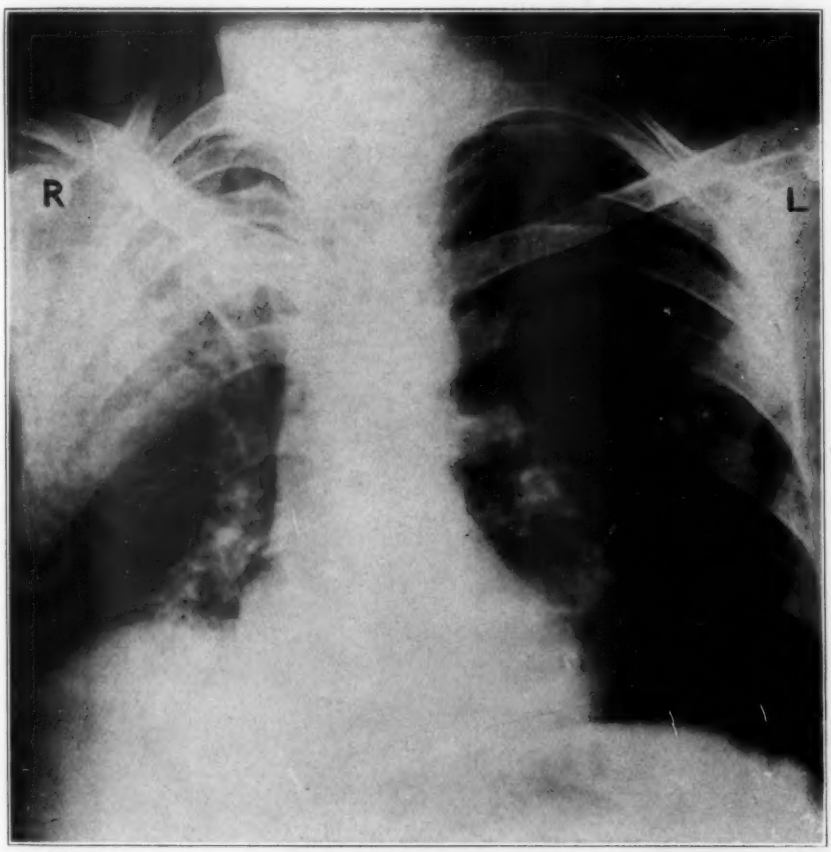

Fig. 10 (R. G.).—Postoperative massive atelectasis, right upper, complicated by pleural effusion, five days after operation; the opacity is limited to the upper and middle lobes, the base is clear; there is cardiac displacement of a lesser degree, but the displacement of the trachea is evident.

the accumulating fluid). As a matter of fact, cases of massive atelectasis will be recognized as they occur, whether postoperative, posttraumatic or associated with inflammatory lesions, when the condition

22. Tidy, H. L.: Acute Lobar Collapse of the Lung, Lancet **1**:1245, 1914.

and its signs are borne in mind as a possibility. The displacement of the heart and mediastinum toward the affected side is the pathognomonic sign. One of the best indicators to suggest this condition is a disparity in the contour and excursion on the two sides of the chest.

Complications and Mortality.—From the febrile course and the mucopurulent sputum, it seems logical to conclude that a pulmonary infection usually accompanies the collapse. When limited to such manifestations,

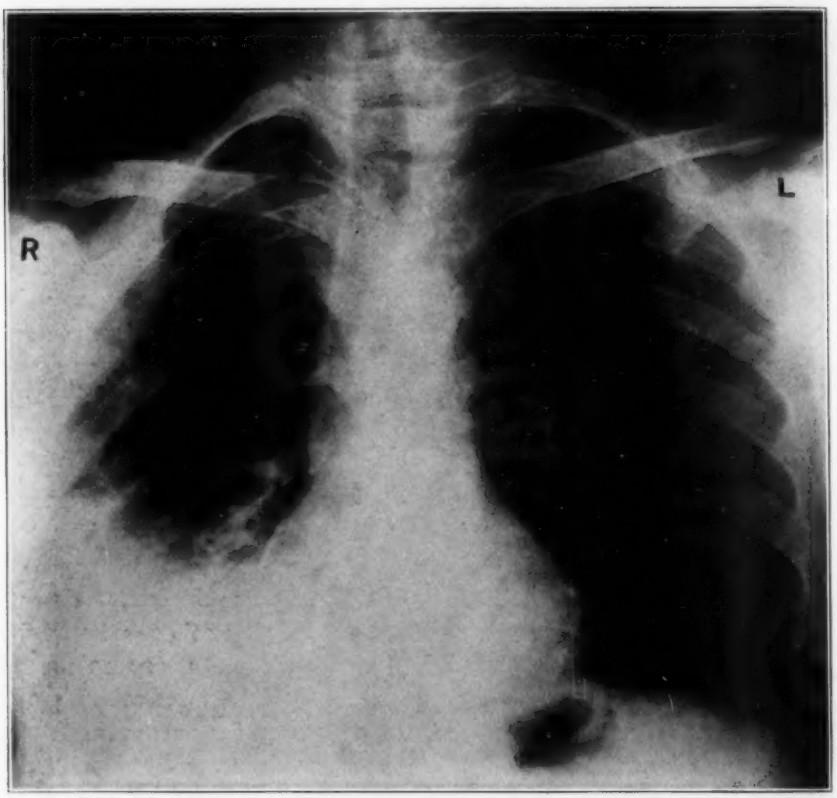

Fig. 11 (R. G.).—Five days after Figure 10 was taken, the opacity of the right upper lobe had largely cleared up, though the trachea was still slightly displaced. Clouding at the right base (in contrast to the clearness in Figure 10) was proved by thoracentesis to be due to pleural effusion.

this associated condition may be merely a bronchitis. One of the striking characteristics of postoperative massive atelectasis is that the large area of pulmonary involvement and the marked cyanosis contrast sharply with the lack of apparent toxicity. A pneumonia patient with the same signs would be critically ill. Scrimger,[16] in the protocol of his Case 21, says, "On the next day after onset he was examined by a physician

who reported that he had the signs of pneumonia at the right base, but did not look like a pneumonia case." This is the usual condition of these patients. In certain instances, however, these cases develop marked toxicity, synchronously with the development of new signs and an increase in the area involved as shown by roentgen ray. This undoubtedly signifies that a widespread bronchopneumonia has occurred as a complication of massive atelectasis.

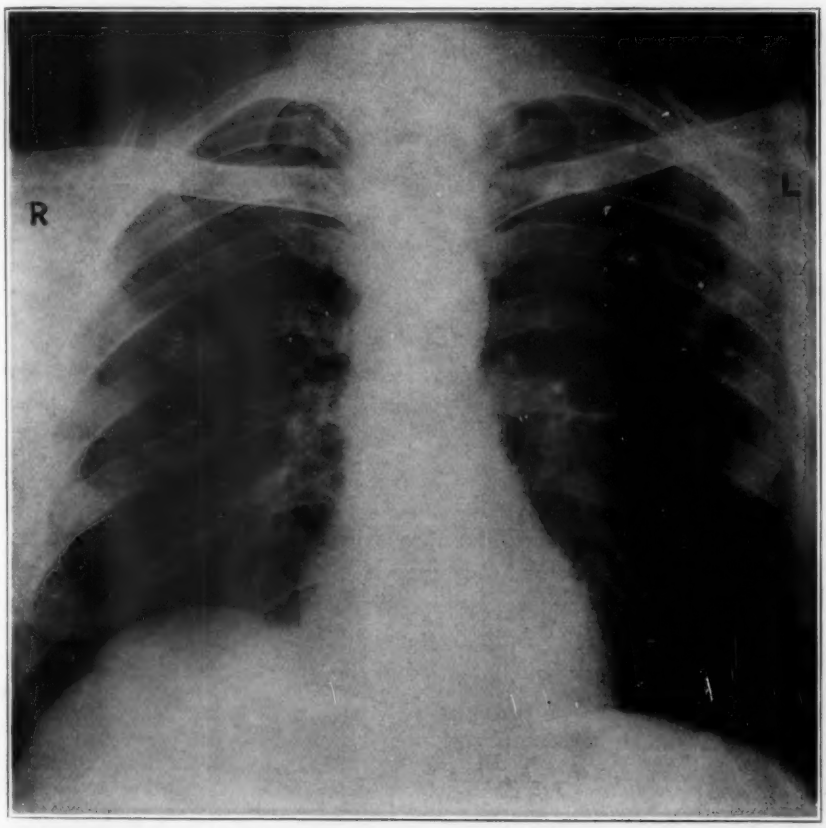

Fig. 12 (R. G.).—Chest one month later than in Figure 11, showing complete recovery.

This course is illustrated by the case of J. L., who Dec. 14, 1923, had a posterior gastro-enterostomy performed for a duodenal ulcer, following which he developed moderate fever, tachycardia and tachypnea with dulness and bronchial breathing at the right base. Figure 8 is a roentgen-ray film made ten days after operation, and, when taken in conjunction with his preoperative film (Fig. 7), proves the diagnosis of postoperative massive atelectasis, though the displacement of the heart was not recog-

nized clinically. The patient at that time did not present any alarming symptoms. Shortly after this, his temperature, which had been showing a moderate elevation, rose sharply to 103.8 F., and he rapidly developed toxic symptoms leading to a stuporous state. Another roentgen-ray examination (Fig. 9) showed the extension of the involvement to the right upper lobe, which had been relatively clear previously. Evidently a confluent bronchopneumonia had arisen to complicate the atelectasis, and he died three days later. Necropsy in this case was unfortunately not permitted. Lee [5] reports a fatal case showing massive atelectasis. In this instance, the patient was operated on under local anesthesia for a strangulated femoral hernia, and developed signs of massive atelectasis three days after operation; two days later, he began to show signs of consolidation of the right upper lobe, to which he succumbed. Necropsy showed a collapse of the right lower lobe with a purulent pneumonia in the two upper lobes. Bronchopneumonia may occur, therefore, as a fatal complication of massive atelectasis.

Pasteur [6] Elwyn [23] and Hirschboeck [24] have recorded instances in which an airless condition at the base of one or both lungs has been found at necropsy. Some of these may have been cases corresponding to the two described above. However, in the absence of the most typical feature of unilateral massive atelectasis, it is uncertain in what way these cases should be classified.

The prognosis of postoperative massive atelectasis is good. In the uncomplicated typical cases that have been diagnosed clinically and reported in detail, the patients have recovered, in spite at times of alarming symptoms. This condition may be complicated by a confluent bronchopneumonia, resulting fatally. Whether there is a bilateral form of massive atelectasis terminating rapidly in death, as Hirschboeck maintains, cannot be definitely settled at the present time. In any event, it appears to occur only rarely.

There is one other complication of massive atelectasis that occurs, viz., pleural effusion. It is probably of infrequent occurrence. Several of the early cases reported in the literature had thoracentesis performed because the dulness and basal clouding suggested fluid, but no fluid was obtained.

The occurrence of pleural effusion as a complication of massive atelectasis is illustrated nicely by the case of R. G. Figure 10 shows the condition, July 12, 1922. A massive atelectasis involving the right upper and middle lobes is present. This distribution of the collapse is unusual but especially fortunate as it is obvious that the base was

23. Elwyn, H.: Postoperative Pneumonia, J. A. M. A. **79**:2154 (Dec. 23) 1922.

24. Hirschboeck (Footnote 17, second reference).

clear, at the time this picture was taken. Five days later, July 17, the upper lobes were largely cleared up, and a basal shadow suggesting fluid was seen; the displacement of the trachea to the right was still evident, showing that the atelectasis had not entirely subsided (Fig. 11). Two hundred cubic centimeters of amber, cloudy fluid was removed, July 21. Figure 12, taken August 16, concludes this series by demonstrating the return of the chest to normal without any residual pathologic condition and with the heart in its normal position.

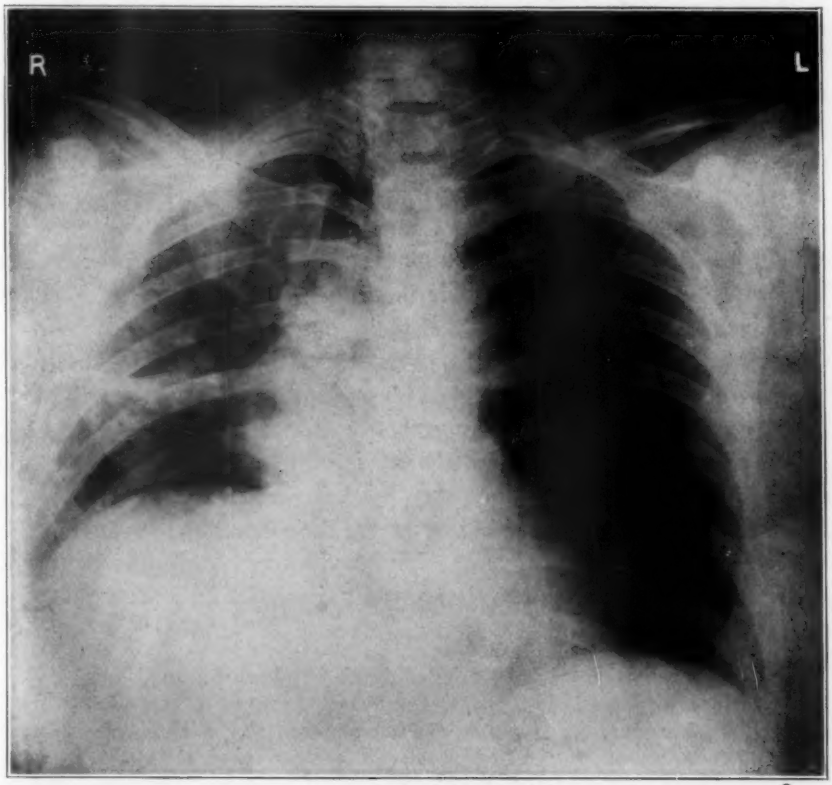

Fig. 13.—Postoperative bronchopneumonia, high right diaphragm, when the temperature was 103 F. and the physical signs in the right chest led to a clinical diagnosis of postoperative bronchopneumonia. The height of the right diaphragm is striking. There is mottling throughout the right lung and a band across the middle of the right thorax. There must be a partial deflation of the right lung, but not massive atelectasis as the heart and mediastinum are not greatly displaced laterally.

Dry pleurisy is also spoken of as a complication of massive atelectasis. It probably does occur, as we know that pleural effusion may. A sound much like that of a pleural friction rub is frequently heard,

most often when the lung is beginning to reexpand. As there are usually many coarse sonorous râles present at the same time, it is difficult, however, to be certain that this is a true friction rub. Empyema has not yet been reported as a complication, but since bronchopneumonia and pleural effusion have been, probably the former will sometime be found.

Pathology.—Postoperative massive atelectasis has been described in the first part of this paper as a clinical entity with symptoms of unilateral collapse of the lung. As an undoubted uncomplicated clinical case of such a condition has not come to necropsy, there is still some uncertainty about the pathology of this condition. The collapse of at least the major part of a lobe has been found at necropsy, in several cases by Hirschboeck, Pasteur and Elwyn, in 1922. In fact, it was the finding of such an atelectasis at postmortem examination in these postoperative cases that first attracted Pasteur's attention to this syndrome.[5] Usually, the condition found was found to be bilateral. Lee's observation in a case complicated by an upper lobe pneumonia confirms the opinion deduced from the signs that there is an atelectasis present, but he does not report the microscopic findings. In this case, he found the bronchus to the collapsed lobe filled with a mucous plug. Until we have more cases clinically diagnosed as postoperative atelectasis which have come to necropsy, it is impossible to be too definite about the pathology of this condition. Leopold,[20] suggests that, in addition to the collapse of the alveoli, there may be an edema of the tissues, designated by him as "drowned lung." As he suggests, direct bronchoscopy of the atelectatic lung would provide instructive data in regard to this.

More pathologic examinations have been made of lungs that show massive atelectasis following war wounds. Bradford describes the appearance of such specimens thus:

> The lung or portion of lung affected is obviously shrunken, heavy, leaden, blue in color and airless. Frequently, it is greatly congested. No obvious gross obstruction of the bronchi is present, and complete massive collapse involving the whole of one lung may be present without any evidence of the existence of any gross obstruction of the main bronchus, such as the presence of a blood clot or foreign body. Obstruction of the bronchus by a blood clot or foreign body is of course capable of producing a condition very similar to that of extensive massive collapse, but there is definite postmortem evidence that such is not the explanation of all cases of massive collapse.

The clinical similarity of massive atelectasis following war wounds and that occurring postoperatively makes it appear altogether likely that the pathologic condition in the latter is essentially the same as this. But no such positive assertion can at present be made.

Treatment.—In the uncomplicated cases, the prognosis is excellent, and the patient will recover after a few days. This is a very fortunate

fact as nothing has as yet been found that will suddenly terminate the pulmonary collapse. Symptomatic treatment for the pain, dyspnea and the annoying cough is indicated, particularly in the form of moderately large doses of morphin (Section III for data on the effect of morphin and other drugs in postoperative massive atelectasis).

To prevent hypostasis, it is probably of distinct advantage for these patients to lie on their side part of the day. As a matter of fact, they

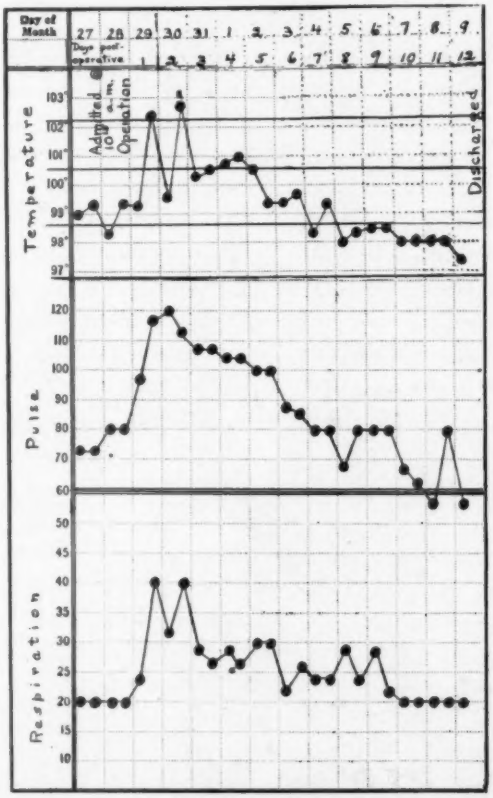

Fig. 14 (Case 37).—Temperature, pulse and respiration record in postoperative massive atelectasis.

involuntarily lie on the affected side for comfort a good share of the time, and they usually cannot lie on the opposite side without developing dyspnea. Consequently, the matter of position will usually take care of itself. The use of blow bottles has been advised on *a priori* grounds, but its value has not been proved. Possibly this procedure may assist after the lung is beginning to expand, but, while it is still collapsed, the effect of the forced ventilation is a greater displacement of the heart and mediastinum toward the affected side: a result not

necessarily advantageous. Oxygen inhalation has been advised also, on account of the cyanosis. This does not seem to be necessary, however, in uncomplicated cases.

If the atelectasis becomes complicated by bronchopneumonia or pleurisy, the treatment should be that indicated for the complication, and will not be reviewed here.

III. ETIOLOGY OF MASSIVE ATELECTASIS

There have been two main theories advanced as to the mechanism of postoperative massive atelectasis. Pasteur,[6] in 1908, observed an airless condition of the lungs in patients dying of peritonitis after operation. This recalled to his mind the fact that he had seen similar conditions in diphtheric paralysis of the diaphragm. It was very natural for him to associate these conditions, and he offered the hypothesis that the collapse of the lung was due to paralysis of the diaphragm and probably of the external respiratory movements. Specifically, he believed that there was no obstruction in the pulmonary air passages.

Elliott and Dingley,[11] in 1914, advanced the alternative hypothesis that the pulmonary collapse was due to obstruction in the bronchial system and absorption of the imprisoned air. They pointed out the fact that, if the bronchus is plugged experimentally in animals, the air is absorbed within a very few hours and massive collapse of the lobes supplied by that bronchus occurs. This was demonstrated by Lichtheim in 1878.[25]

In arriving at a conclusion about this matter, there are two separate groups of phenomena which must not be confused. In the first instance, there are the factors governing the condition that has been described in the preceding section of this paper as a clinical entity, postoperative massive atelectasis. The second condition is less definitely characterized clinically than the foregoing but is probably of great importance in the postoperative pulmonary complications and may be the forerunner of the foregoing clinical entity. It is a relatively airless condition of part of the lungs occurring in a very pronounced fashion in some cases, and quite possibly, as Briscoe[26] maintains, to some extent in all postoperative patients, owing partly to the supine position.

I shall now consider the mechanism concerned in the first of the conditions, namely, massive atelectasis. It seems impossible that the explanation offered by Pasteur and subsequently advocated by several of

25. Lichtheim, L.: Versuche über Lungen Atelektase, Arch. f. exper. Path. u. Pharmakol. **10**:54, 1878-1879.

26. Briscoe, J. C.: The Mechanism of Postoperative Massive Collapse of the Lungs, Quart. J. Med. **13**:293 (April) 1920.

the other investigators in this field can adequately explain all the signs and roentgen-ray manifestations of this condition, both on certain *a priori* grounds and from clinical observations. Probably the most striking single phenomenon connected with postoperative massive atelectasis is the displacement of the heart and the mediastinum. As a matter of fact, the partition separating the pleural cavities serves as an

Fig. 15 (Case 37).—Postoperative massive atelectasis, three days after appendectomy; heart and trachea displaced to the right. At this time, the patient had fever, tachycardia, dyspnea, cough, pain in the chest, cyanosis, depression and immobility of the right thorax, dulness and suppressed breath sounds, but no râles.

excellent mechanism to demonstrate relative differences of pressure in the two pleural cavities. Obviously, it is not as delicate as a recording tambour, but acts on the same principle, provided that it has not been rendered fixed by a chronic inflammatory process. The acute displace-

ment of the heart and trachea to the collapsed side, then, must indicate a relative lowering of the pressure within the affected pleural cavity as compared with the opposite side.

By unilateral paralysis of the diaphragm or collapse of the chest wall, the negative pressure in this pleural cavity would be reduced because it is maintained normally by these mechanisms. Therefore, by this means no increase in the negative pressure would be obtained. And, provided the air passages are freely open, any difference in the pressure of the two sides would be equalized, as it is obviously easier to allow a slight exchange of air within the lungs than to displace with considerable resistance the mediastinum. Thus, on *a priori* grounds, it seems inconceivable that paralysis of the diaphragm or external respiratory mechanism alone could produce a displacement of the mediastinal structures toward the affected side.

This supposition is amply confirmed by clinical and experimental data. Crymble [27] records a case of war injury in which there was complete paralysis of the external respiratory muscles on both sides accompanied by paralysis of one phrenic nerve. The patient had only one half of the diaphragm remaining with which to carry on respiration. According to the theory of Pasteur, this should therefore have produced massive collapse of the lung on the side of the phrenic paralysis. The resulting condition was interesting. The diaphragm on the paralyzed side was very high, producing a most unusual roentgenographic picture, but the heart shadow was not displaced laterally. Elliott and Dingley [11] produced, in cats, a paralysis of the respiratory mechanism on one half of the body by hemisection of the cord. By this means, this theory was tested, and no displacement of the heart resulted, in spite of the fact that respiration was unilateral. In fact, in Pasteur's own article in 1914,[10] he records the details of a postdiphtheric case in which there were symptoms of unilateral paralysis of the diaphragm on the right side, and he states definitely that the heart was not displaced. Furthermore, there are definite cases of postoperative massive atelectasis that affect chiefly the upper lobe (Fig. 10), and, even in the usual type with basal involvement, roentgen-ray study of the early stages in two of our recent cases, Cases 38 and 40, has shown opacity in the midzone while the supradiaphragmatic zone was still air-containing (Figs. 10 and 19). These facts would be difficult to explain solely as the result of diaphragmatic paralysis. It seems, therefore, that paralysis of the muscles of respiration cannot constitute the whole cause of the postoperative massive atelectasis, and that there must be present, either accompanying such a paralysis or entirely independent of it, some obstruction in the air passages serving the affected area.

27. Crymble, P. T.: Gunshot Wounds of the Chest, Brit. J. Surg. **5**:363 (Jan.) 1918.

What is the nature of this obstruction? With the postoperative form that has been described, massive atelectasis is accompanied by a fever and usually there is mucopurulent expectoration within a day or so after the onset. This suggests a plugging of the bronchi to the affected area by an inflammatory exudate. It appears probable that bronchitis usually does accompany the massive atelectasis and the tenacious secretion quite probably does assist materially in the obstruction to the involved lung. It does not seem, however, that this explains the onset of the condition satisfactorily. If the condition were merely a diffuse bronchitis with obstruction to the air passages, we should expect to find its manifestations bilateral, whereas the striking factor of the condition is the unilateral distribution of the atelectasis. Again, the onset of the condition is sometimes so sudden that it is extremely difficult to conceive of its arising from an inflammatory cause. For example, Bradford [12] quotes the case of a soldier who received a very superficial wound of the chest wall without penetration or fracture of the ribs. The man walked 4 miles to a dressing station where the bullet dropped out of the wound on examination. He was sent back to the base hospital complaining of no symptoms. Yet, the same night, it was found that he had a contralateral massive atelectasis. Also, postoperatively the condition may follow local or spinal anesthesia. It would be difficult to explain this on the basis of infection. I believe, therefore, we are justified in concluding that there is some reflex mechanism which produces, either directly or secondarily by secretion, an obstruction to the air passages supplying part of the lung. But it is quite certain that the atelectasis is not caused by complete obstruction to the main bronchi, as loud tubular breathing is very often heard in the affected area. Scrimger has alluded to the interesting experiments of Carlson and Luckhardt [28] in which the latter produced contraction of the saclike lung in amphibia by electrical or mechanical stimulation of the nasal passages, small and large bowel, kidney, bladder, etc. I know of no work which proves such a mechanism in mammalia, but the analogy is nevertheless interesting. It is certain that if the air passages in a large area of one lung are obstructed, massive atelectasis of the area affected will result with displacement of the mediastinum. This has been proved clinically in cases of bronchial tumor, and by experimental obstructions in animals.

In this connection, certain observations made in our cases seem to throw some additional light on the mechanism of postoperative massive atelectasis. Patient 39, at the time of my first examination, showed loud

28. Carlson, A. J., and Luckhardt, A. B.: Studies on the Visceral Sensory Nervous System, IV, V, VI and VIII, Am. J. Physiol. **55**:13 (Feb.), 31 (Feb.), 212 (March); **56**:72 (May) 1921.

sonorous râles throughout both chests. After examination of the anterior chest, the paitent was turned on his left (unaffected) side. Within about three minutes, he developed marked dyspnea, although he had not been conscious of any respiratory distress while on his back. He was therefore allowed to return to his back, and, about five minutes after the original examination, his chest was again auscultated and at

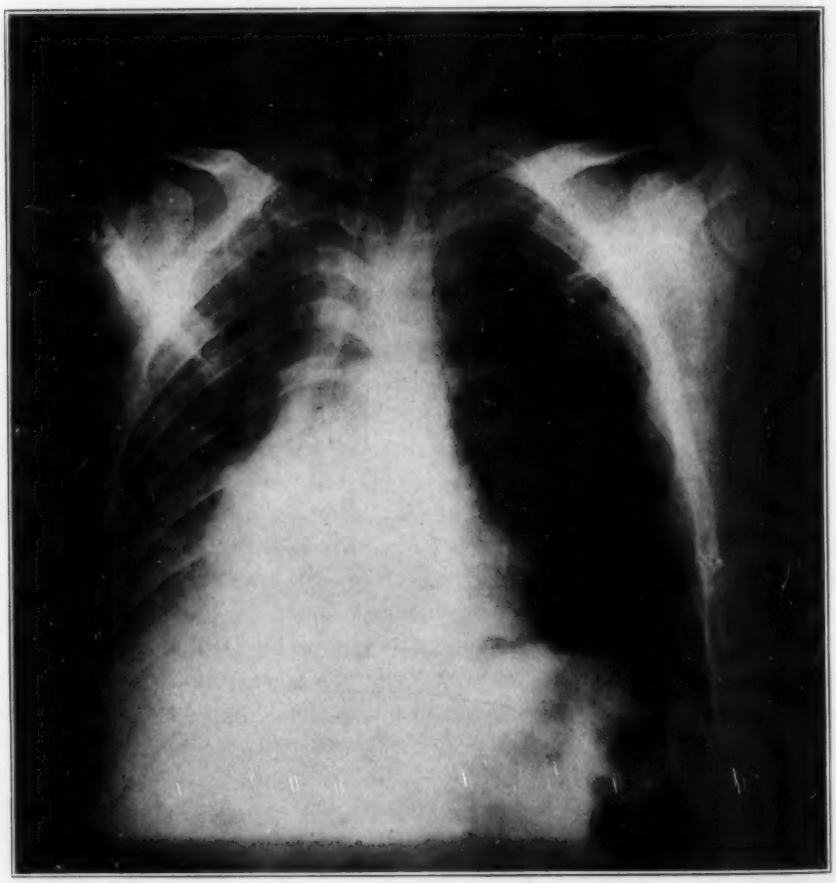

Fig. 16 (Case 37).—Six days after operation, the opacity beginning to clear; displacement of heart and mediastinum unchanged; respiratory excursion of right chest returning; râles have just appeared.

no point were any râles audible, either on the affected or the unaffected side.

This unexpected, rapid alternation in the presence and absence of râles was observed also in Case 38. Musical râles were present in abundance throughout both sides of the chest when examined,

January 28. One-half hour later, when the examination was repeated, there were only a very few râles present on the affected side, and none on the opposite side. One other fact seems to confirm the belief that there is some mechanism producing bilateral pulmonary involvement, presumably of a reflex nature. Measurement of the vital capacity showed a diminution greater than should be expected from complete suppression of the functions of one lung. The vital capacity of Patient 38, for example, was 25 per cent. of normal. This great reduction in the available respiratory reserve is also demonstrated forcefully by the marked dyspnea that is caused in these patients by the least exertion, such as moving about in bed.

From these observations, it seems that there must be some reflex which causes a constriction in the air passages, probably affecting the small bronchioles not dependent originally on infection, and acting on both lungs to some extent. Probably the chiefly unilateral manifestation of the signs is due to a difference in the degree to which the two sides are affected. We have no data as to the exact channel through which this reflex acts. There seem to be three possibilities: (1) vasomotor; (2) bronchiole spasms, (3) swelling of the mucous membrane.

1. Vasomotor. Dilatation and stasis in the blood vessels could produce obstruction in the bronchioles by an outpouring of secretion. Our present knowledge of the pulmonary vasomotor control gives no absolute proof of such a mechanism causing a maximal effect in one lung. After producing a unilateral closed pneumothorax in monkeys, Yates [29] has reported certain sudden, spontaneous variations in the intrapleural pressure on one side. He interpreted these phenomena as resulting from changes in the distribution of blood within the pulmonary circulation. Also, the distention of the lung in amphibia by sensory stimuli is thought to result from a pulmonary vasomotor reflex, according to the work of Carlson and Luckhardt.[28]

2. Bronchiole spasm. This naturally suggests itself as a possibility on account of the sonorous râles which have been found to vary from minute to minute and are present in both lungs. However, our knowledge of bronchiolar spasm shows that it produces exactly the reverse condition, namely, emphysema with a bilateral distribution. Unless, then, we are dealing with a new type of reaction, this hypothesis seems unlikely.

3. Swelling of the mucous membrane. The third possibility is an edema of the mucous membrane lining the bronchioles, similar possibly

29. Yates, J. L.: Effects of Acute and Chronic Pneumothorax, Am. J. M. Sc. **165**:1 (Jan.) 1923.

to angioneurotic edema. We suggest this in view of Hoover's [30] recent discovery in regard to the mechanism in anaphylaxis of the guinea-pig. He has shown that the bronchial obstruction is due to swelling of the mucous membrane, and not to bronchial spasm. Further investigation will be required to determine which of these is the effective mechanism

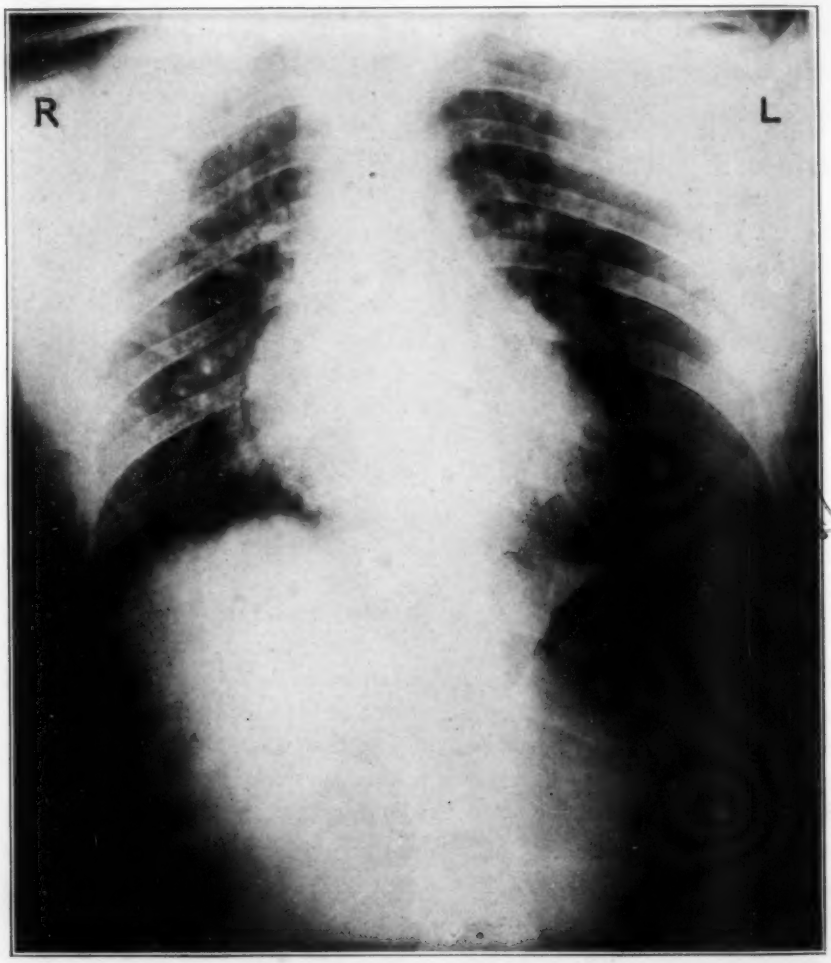

Fig. 17 (Case 37).—Nine days after operation, symptoms have disappeared, except the cough; the heart is returning to normal position.

which reflexly causes the obstruction present in massive atelectasis. On the basis of our present knowledge, it seems most likely that this is vasomotor.

While the exact nature of its onset is uncertain, after the collapse has occurred, there seems to be a low grade type of infection set up.

30. Hoover, C. F.: Personal communication to the author.

This is quite in keeping with the effect of obstruction in any of the systems leading to the exterior of the body. With this bronchitis, tenacious secretions are poured out into the bronchioles and undoubtedly assist in maintaining the obstruction. Quite possibly, mucopurulent material may be entirely responsible for the continuance of the atelectasis. The sudden complete relief of symptoms occurring after a coughing spell reported in some cases suggests this possibility, as in Cases 15 and 16 of Scrimger. Although, as outlined above, it seems impossible for paralysis of the diaphragm alone to cause massive atelectasis with displacement of the heart, quite probably it also assists in maintaining the atelectasis. Many observations of a high immobile diaphragm have been made in massive atelectasis. In Case 40, a recent typical case, after the symptoms, signs and roentgen-ray opacity had disappeared, paradoxic respiratory movements of the diaphragm on the side that had been involved were seen; so that a month after operation unilateral paresis of the diaphragm was present as the only residual sign.

In order to throw any light possible on the mechanism involved, a study was made in massive atelectasis of the pharmacodynamic action of certain drugs which might affect the reflex involved. The topographic location of the operations that are followed by massive atelectasis is such as to suggest that either the vagus or the sympathetic system is involved as the conduction path of the reflex. These conditions follow almost exclusively operations and injuries to the abdomen and chest. They have never been recorded after operative or traumatic lesions of the extremities alone. These drugs which suggest themselves in this connection have been tested, viz., (1) atropin, (2) epinephrin and (3) morphin.

1. Atropin. In Case 37, December 31, three days after operation and at the time of the height of his symptoms, the exact position of the heart was determined on the fluoroscopic screen, and it was revealed that its location did not change on successive views during a period of fifteen minutes. Atropin sulphate was then given subcutaneously while the heart was being observed fluoroscopically. After 1.75 mg. had been given in two doses within twenty minutes, a physiologic effect became manifest by the cessation of perspiration, which previously had been very abundant; there was no change in the position of the heart. Seventy-five hundredths milligrams more, making in all 2.5 mg. within thirty minutes, was administered subcutaneously. Within a minute, the heart, the position of which had remained unchanged on repeated observations over a period of forty-five minutes, rather surprisingly shifted more than a centimeter to the right (toward the collapsed lung), and the patient developed an increased dyspnea and began to cough. On account of the exacerbation of symptoms, the observation had to be

terminated, and the patient was returned to his bed. The increased respiratory distress lasted for over an hour, and gradually subsided. The pulse rate before the administration of atropin was between 110 and 120. After the successive doses of atropin, the pulse was not counted in the fluoroscopic room, but it did not appear to be greatly changed, and it was 120 at the close.

From these data, it seems clear that at least after the atelectasis is established, atropinization does not overcome the bronchiolar obstruction, but if it produces a definite effect, this tends to increase the degree of collapse. This result certainly does not lend support to the theory that the reflex that initiates the massive atelectasis acts through the vagus by bronchioconstriction.

2. Ephinephrin. Atropin having failed to relieve the condition, the effect of epinephrin (0.5 c.c. of 1:1,000) given hypodermically was tried. This was not done under the fluoroscope. But it, too, failed to produce striking changes in the physical signs, or alleviate the symptoms permanently. In Case 38, the epinephrin seemed temporarily to relieve the cough which was constantly annoying the patient before this drug was administered. The three cases so far tested have shown a definite hypersensitiveness to epinephrin. In each instance, this test was carried out in the febrile or convalescent period and not preoperatively. It is quite possible, therefore, that these patients merely show the same hypersensitiveness that Peabody, Sturgis, Tompkins and Wearn [31] have shown occurs frequently with various infections. On one of the patients, Case 40, the test was repeated two weeks after discharge when he was still hypersensitive.

3. Morphin. This narcotic was given hypodermically in rather large doses, 30 mg. to Patient 38, January 28 and 29; roentgen-ray plates being taken before and twenty minutes after administration. The patient, who was having much distress and dyspnea before the morphin was given, was symptomatically much relieved by it and dozed off with less labored respiration. There was no measurable alteration, however, in the position of the heart.

In all the observations on the effect of drugs in massive atelectasis, the fact must be borne in mind that, at the time of the exhibition of the drug, the atelectasis was well established. The possibility still remains that at the onset the condition might have been affected differently. Probably this point will not be entirely settled until massive atelectasis can be produced at will in animals by a reflex similar to the one acting

31. Peabody, F. W.; Sturgis, C. C.; Tompkins, E. M., and Wearn, J. T.: Epinephrin Hypersensitiveness and Its Relation to Hyperthyroidism, Am. J. M. Sc. **161**:508 (April) 1921.

in the postoperative and traumatic cases. So far, no successful attempt to do the latter has been reported. The outstanding features of these observations of the pharmacodynamic effects in massive atelectasis are: (1) So far, the patients have possessed a hypersensitiveness to epinephrin; (2) atropin to a physiologic effect does not relieve the displacement of the heart, and (3) morphin produces some symptomatic

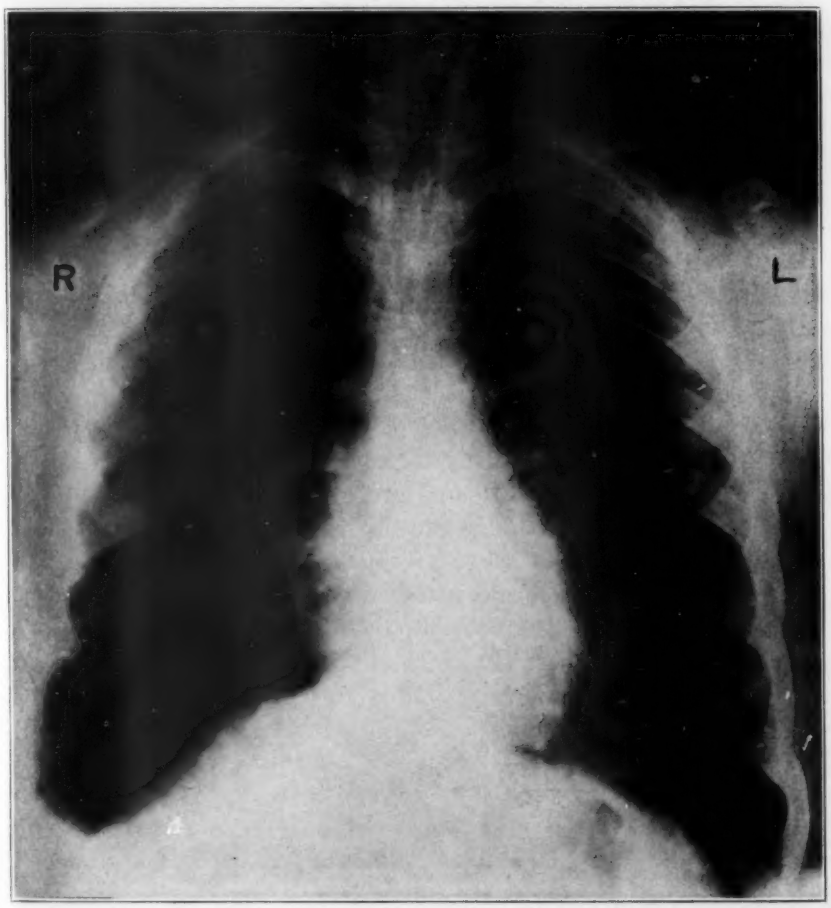

Fig. 18 (Case 37).—Twenty-four days after operation, the chest has completely cleared up. The patient went back to work, symptomless.

relief without greatly altering the physical condition of pulmonary collapse.

In regard to the clinical entity that we recognize as postoperative massive atelectasis, it seems reasonable to conclude that it is caused by an obstruction to the respiratory air passages with subsequent absorption

of the imprisoned air, thus creating a relative difference in the negative pressure within the two pleural cavities. Presumably, the obstruction is produced by a reflex stimulation which affects both sides, but quite unequally. Unilateral paresis of half of the diaphragm may be another outlet of this reflex. This condition is an interesting and important clinical entity. However, it seems probable that the postoperative factors producing pulmonary collapse are, in other less obvious relationships, of even greater significance than the striking and dramatic clinical picture that they produce in massive atelectasis.

IV. ATELECTASIS AS A FACTOR IN POSTOPERATIVE PULMONARY COMPLICATIONS

A mass of data is accumulating which does not yet prove, but does strongly suggest, that collapse of lung tissue is an important element in most postoperative pulmonary complications, except large fatal pulmonary emboli. Elwyn,[23] in 1922, concluded that most of the cases of postoperative pneumonia were due to infection taking place in atelectatic areas. In 1924, he reported further data in this matter. Many cases of postoperative bronchopneumonia showed signs of consolidation which were cleared up after a paroxysm of coughing, and, in some, a high diaphragm was observed.

As is shown in the examination of cases of postoperative pulmonary complications, there seem to be varying degrees of collapse of the lung. In the dramatic form, this culminates under the proper condition in the striking clinical picture of massive atelectasis. In other cases, the displacement of the heart and mediastinum can only be demonstrated by a careful comparison of the roentgen-ray film taken at the time of the complication with the normal picture of that individual. In still other instances, the diaphragm on one side is greatly elevated, but the heart is not greatly displaced. Figure 13 illustrates a marked example of this. The right diaphragm is very high, but the mediastinum is about in its normal position. This, then, does not correspond to the clinical entity "massive atelectasis," but there must obviously be a relatively airless condition of the right lung base. Paresis of the diaphragm on one or both sides, as determined fluoroscopically, is frequently seen in postoperative bronchopneumonia cases. And, furthermore, two examples have been referred to in the section on complications, in which typical massive atelectasis of the base was complicated by bronchopneumonia of the upper lobe. Also, in cases of massive atelectasis following war wounds, purulent bronchitis and pneumonia limited absolutely to the collapsed lung were found.[2] So that the clinical evidence is strongly suggestive of some etiologic relationship between the collapse and the pneumonic process in the lung.

There is also a striking parallelism in the distribution of postoperative massive atelecatsis and postoperative bronchopneumonia. In abdominal operations, they are both common. The latter was seen only once in 132 operations on the extremities by Elwyn.[4] Bradford[12] informs us that massive atelectasis was not seen in the war wounds after the extensive injuries to the extremities, and none of the postoperative cases on record have followed surgery limited to the extremities.

However, a relatively collapsed area of the lung does not seem to be the only condition necessary for the production of postoperative pneumonia. Pasteur[10] reported the fluoroscopic examination of four uncomplicated appendectomy cases. One of them showed paresis of the diaphragm, but did not develop any symptoms. Briscoe[26] found evidence of deflation of both lower lobes occurring commonly after abdominal operations, and believes that some degree of it invariably follows a restrained supine position. Briscoe, Hirschboeck, Pasteur, Elwyn and others have recorded the finding of deflated lobes at necropsy without pneumonia. Further, Bradford records afebrile cases of massive atelectasis following war wounds. Obviously then, some other condition in addition to atelectasis is required for the production of a postoperative bronchopneumonia. Elwyn[4] assumes that this second factor is a preexistent infection in the lung, or irritation by the anesthetic. Infection surely must occur, but, as the usual determining factor, this explanation seems to us unsatisfactory for the same reasons that Cutler and Hunt[1] and Elwyn[4] rejected it as a complete explanation for the occurrence of postoperative bronchopneumonia which follows local anesthesia just as it does general anesthesia and for which precedent pulmonary infection is not essential. The causal relationship of hypostasis for the production of bronchopneumonia in the aged is a fact that no one doubts. This suggested to us that the second factor might well depend on the local circulatory condition. Cutler and his co-workers[1] have presented convincing data suggesting the importance of small emboli from the operative field as an etiologic factor in the production of postoperative pneumonia. Karsner[32] demonstrated that small experimental emboli in the dog's lung produced only temporary circulatory changes. However, if the venous return was obstructed or the lung was collapsed by injecting olive oil into the pleural cavity previous to the embolism, true infarcts were produced. This suggests a logical combination of infarction and collapse in the production of postoperative pulmonary complications. We, therefore, propose as a working hypothesis, that the lodging of a small embolus from the operative site in an atelectasis area is the usual mechanism producing postoperative bronchopneumonia. In certain cases

32. Karsner, H. T., and Ash, J. E.: Studies in Infarction: II, Experimental Bland Infarction of the Lung, J. M. Res. **22**:205, 1912-1913.

of massive atelectasis, the expectoration is definitely blood-streaked, suggesting that this combination of infarction with the collapse of lung tissue does occur.

CONCLUSIONS

1. Postoperative massive atelectasis presents a striking clinical syndrome, and is relatively frequent.

2. It is differentiated from all other acute conditions by displacement of the heart and mediastinum toward the side that shows evidence of pulmonary consolidation.

3. Asymmetry of the chest, unilateral limitation of respiration, and cyanosis are early and striking signs.

4. Latent cases occur which are nearly symptomless, and others are so severe that they resemble pulmonary embolism.

5. The prognosis is excellent. Massive atelectasis may be complicated by bronchopneumonia, and in this group the fatalities occur.

6. The mechanism of massive atelectasis appears to be a reflex blocking of the finer air passages in the affected lung tissue, quite possibly of vasomotor origin.

7. Collapse of lung tissue after operation seems to be a significant element in other pulmonary complications. Infarction in an atelectatic area is suggested as the usual mechanism of postoperative bronchopneumonia.

REPORT OF CASES

CASE 37.[33]—A vigorous young man, aged 18, single, who entered the hospital, Dec. 27, 1923, with the complaint of pain and tenderness in the right lower abdomen, was given the preoperative diagnosis of recurrent appendicitis. He had had four attacks, all of which had been comparatively mild. He had recovered from the last of these, three days before admission. There had been no symptoms referable to the cardiorespiratory system in the past history.

Physical examination, on admission, was negative except for slight residual tenderness well localized in the right lower quadrant of the abdomen. The thorax was well formed and symmetrical; expansion was good, and equal on both sides. In the lungs, the percussion note was resonant throughout. Breath sounds were vesicular. There were no râles. In the heart the point of maximum impulse was seen and felt in the fifth interspace, 8.5 cm. to the left of the midsternal line. The left border of dulness was 8.5 cm. to the left of the midsternal line. The right border of dulness was not outside the right sternal margin. Sounds were regular and of good quality. The second pulmonic sound was greater than the second aortic sound. They were no murmurs or thrills. The pulses were equal, regular, synchronous and of good volume and tension. The vessel walls were not felt. The blood pressure was 120 systolic, 78 diastolic. The temperature was 99 F.; the pulse, 70-80; respiration, 20. The white blood cells were 10,300.

An operation was performed, December 28, the day after admission, and consisted of a simple appendectomy with some mobilization of the lateral wall of the ascending colon and an exploration of the upper abdomen. The anesthetic was ether, and the anesthesia was uncomplicated. The anesthetist noted cough and

33. This patient was demonstrated before the Harvard Medical Society, Jan. 8, 1924.

mucus during induction with an uneventful subsequent anesthesia. The bedside notes show that the patient began to have a slight cough two hours after operation, and that during the afternoon he complained several times of pain on coughing. At midnight, thirteen hours after operation, he complained of severe pain in the right thorax with respiration. The cough troubled him sufficiently during the first night, so that he was twice given medicine for it. December 29, the first postoperative day, he was troubled greatly with cough and pain in the right chest. His temperature, pulse and respiration mounted sharply in the afternoon (Fig. 14). There was no expectoration, but there was marked respiratory distress. Examination by the intern, who thought the patient was developing postoperative pneumonia, showed relative dulness at the right base, with suppression of the breath sounds over the dull area. No râles were heard. The position of the heart was not determined at this time. The second postoperative day, December 30, the symptoms continued with increased severity. The only position that the patient would assume was that of lying on his right side, and there was a definite cyanotic hue. My examination of the chest revealed: in the lungs, on the right there was depression of the infraclavicular fossa and marked limitation of respiratory movement, and relative dulness throughout except in the supraclavicular fossa, with suppression of breath sounds in the dull area. At the right base anteriorly, the impaired percussion note had a wooden, tympanitic timber. At the right base posteriorly, the note was flat, and breath sounds were inaudible. On the left, the respiratory excursion was great, the percussion note hyperresonant and the breath sounds harsh but vesicular in character. No râles were heard on either side. In the heart, the superficial cardiac dulness was completely obliterated. The apex beat was not felt. The relative cardiac dulness could not be determined on the right; on the left, it seemed to be 3 cm. to the left of the midsternal line. Heart sounds were clear and without murmurs. There was no abnormality of the pulse except that the rate was 120. The liver edge could not be palpated. The diagnosis of postoperative massive atelectasis was corroborated by roentgen ray, which showed diffuse clouding of the right lower lung and the displacement of the heart and mediastinum to this side (Fig. 15). The patient's symptoms reached their maximum on this day. He was persistently troubled with an unproductive cough and pain in the chest, requiring three doses of morphin, and the temperature reached its maximum of 102.8. The leukocyte count was 23,300. However, in spite of the patient's marked discomfort, the cyanosis, tachycardia and very extensive pulmonary dulness, a striking feature of the case was the fact that he did not appear toxic.

After the second postoperative day, the symptoms and signs began to recede gradually. On the third day, the heart, while still displaced, had begun to return to its normal position, and the dulness anteriorly had diminished. Still there were no râles. The temperature gradually fell, and remained normal after the seventh day (Fig. 14). The respiratory excursion of the right thorax was greatly restricted for four days. January 3, the sixth postoperative day, paroxysms of cough still occurred, though these were much less frequent, and adventitious sounds were heard for the first time. These consisted in sibilant and sonorous râles, and were heard on both sides of the chest, being more marked on the right. The heart was still displaced somewhat to the right, as shown by percussion and roentgen ray (Fig. 16).

Two days later, January 5, the musical râles were so marked that they could be heard from the bedside. These wheezes were throughout both lungs. Twelve hours later, January 6, no adventitious sounds could be heard anywhere on careful examination of the chest, and they were not found on subsequent examinations. Roentgen-ray examination, on the latter day (Fig. 17), showed only

slightly greater density on the right than on the left; also, the heart and trachea were back nearly to their normal position. The patient was still having occasional coughing spells. He was discharged from the hospital symptomless, twelve days after operation. Discharge examination of the chest was normal. He returned by request, January 21, three and a half weeks after operation. He had had no symptoms, and was back at full work. Physical and roentgen-ray examinations of the chest were quite normal (Fig. 18).

CASE 38.—A man, aged 20, a robust laborer, who entered the hospital, Jan. 23, 1924, six years previously had had an appendiceal abscess drained through a McBurney incision, leaving him with a rather large ventral hernia in the wound; and since then he had had attacks of pain in the right lower quadrant, accompanied by dysuria. He had never had any symptoms referable to the cardiorespiratory systems. Examination was negative except for the post-operative hernia and tenderness below McBurney's point. Heart and lungs were found normal. The temperature was normal. The pulse was 70. The respiration was 20. The urine was negative. The white blood cells were 6,700.

An operation was performed, January 26, consisting of appendectomy and repair of the ventral hernia. The anesthetic was ether, and was well taken. Hiccough, during the first part of the operation, was noted. His immediate postoperative recovery was uneventful. The next day, he complained of a great deal of pain in the incision, and, during that afternoon, his temperature, pulse and respiration mounted sharply, the temperature reaching the maximum of 101 F.; the pulse, 120, and the respiration, 52. He complained of no symptoms referable to the thorax. Examination at 8 p. m. showed that the lips had a definite cyanotic hue. With the patient on his back, the left chest was more prominent than the right. The respiratory excursion of the right chest was definitely limited, and was estimated as half that of the left. The whole of the left chest was hyperresonant. There was impairment of the percussion note at the right base. Posteriorly, this was well marked up to the sixth rib. Breath sounds were distant and bronchovesicular in quality over the dull area. Sibilant râles were heard over both chests in abundance at one time, while half an hour later there were none audible over the left chest and only occasional scattered râles over the right side. The exact position of the heart was difficult to determine on account of the hyperresonance on the left and the dulness in the right. The left border seemed to be about 2.5 cm. inside the left nipple. The superficial cardiac dulness was completely obliterated. There was no abnormal pulsation seen to the right of the sternum. It was my impression, at this time, that this was a massive atelectasis of lesser degree, and the roentgen-ray examination verified the slight displacement of the heart to the right. There was clouding of the middle part of the right chest. Interestingly, the extreme base was clear (Fig. 1). The next day, January 28, the physical signs had progressed remarkably. With the patient on his back, there was a striking depression of the right thoracic wall with a narrowing of the intercostal spaces (Fig. 5). The right chest moved scarcely at all with respiration, while the left side had an increased excursion. The area of dulness had increased. On the right, flatness now began at the fifth rib in front and at the ninth rib behind, and relative impairment extended throughout the whole back. There was loud, tubular breathing posteriorly on the right from the third to the ninth ribs. This was accompanied by marked bronchophony and pectoriloquy. Below this area, the breath sounds were completely suppressed, and at the apex they were bronchovesicular. There were no râles heard except a few nonpersistent crackles anteriorly. Abnormal cardiac pulsation could easily be seen in an area extending 3 cm. to the right of the sternal border in the second, third and fourth interspaces, and the heart sounds could be heard best just to the left of the sternum.

A roentgenogram taken this day, twelve hours after the previous one (Fig. 2), showed an increase in the clouding of the right lung; and, in particular, the clear zone at the extreme base was now entirely obscured. The heart and mediastinum were displaced further to the right than on the previous day.

The clinical course was a rather rapid amelioration of symptoms and clearing of signs. The temperature reached its maximum of 103.4 fifty-two hours after operation, and remained elevated for two more days, after which it was not above 99. The white blood count was 14,200, January 28, and was still at this figure, February 2. The respiratory rate was constantly elevated for seven days after operation, and did not remain below 25 until the eleventh day. February 4, nine days after operation, there was still a definite impairment of the respiratory excursion on the right side and some residual dulness at the base. Examination before discharge, February 11, sixteen days after operation, revealed no residual signs in the chest.

Case 39.[34]—A vigorous young man, aged 19, who came into the Boston City Hospital, Jan. 24, 1924, with acute appendicitis, had had a chronic cough, productive at times, for four years. Two weeks before admission, he had pain in the chest accompanying this cough, and, on admission, the patient had paroxysmal attacks of coughing. On examination before operation, he had diffuse coarse râles throughout both chests; otherwise the findings were normal. On account of his cough and râles, a roentgenogram was taken before operation which showed some mottling above the right hilum, but was otherwise negative (Fig. 3). The temperature was 99.8 F.; the pulse, 100; respiration, 25. The white blood cells were 20,000.

Appendectomy was performed at once, the anesthesia being nitrous oxid-oxygen-ether sequence. The appendix had ruptured, forming a small abscess. Consequently, drainage was necessary. The patient did very well after operation, and complained of no unusual symptoms. His temperature was elevated, reaching 102 the day after operation. This amount of reaction was not surprising in view of the appendiceal abscess that had been present. Two days after operation, the front of the chest was examined by a medical consultant on account of the preoperative roentgen-ray findings that suggested a latent tuberculous focus. The examiner reported a dextrocardia. This suggested at once the diagnosis of postoperative massive atelectasis, which was verified by examination and roentgen ray (Fig. 4). The patient had not complained of any respiratory symptoms, and his fever had been ascribed to the appendiceal abscess. If it had not been for the fortuitous examination of the chest, this complication would probably not have been discovered. The physical signs were typical. There was depression of the thoracic wall and marked limitation of respiratory movement on the right side, with dulness at the base beginning at the fifth rib in front and at the third rib behind. The breath sounds over the dull area were very distant. There were loud wheezing râles heard over both chests. He had no dyspnea while lying on his back; when turned on his left side, he was perfectly comfortable at first, but, within three minutes, he developed marked dyspnea and a definite cyanotic tint to the lips. Both these signs disappeared when he resumed the previous dorsal position. The left lung was hyperresonant. The superficial cardiac dulness was obliterated, and, by percussion, the left border seemed just to the left of the sternum.

The patient was quite comfortable throughout except for occasional cough and some mucopurulent sputum. By February 2, the heart had returned to its normal position, and the lung signs had cleared up. Roentgen-ray examination

34. It was through the courtesy of Dr. J. C. Hubbard of the Boston City Hospital that I had the opportunity to study this case.

on that date showed only a residual band of clouding in the region of the right middle lobe. The patient got up and was about the ward, February 6, but felt weak and had a persistent cough, productive of thick yellow sputum in which no tubercle bacilli could be found. However, February 10, he coughed up 120 c.c. of bright red blood. He had no further hemoptysis; the cough and sputum diminished, finally disappearing about February 26. He was discharged, March 10, symptom free and without signs except for some impairment at the right apex..

CASE 40.—A vigorous man, aged 26, who entered the hospital, April 27, 1924, on account of an attack of acute appendicitis of two days' duration, which was subsiding, about the time of the onset, had eaten some strawberries, two hours after which he developed urticaria. The patient had had one previous attack of acute appendicitis eight months before, lasting four days. He had never had any cardiorespiratory symptoms. On admission, physical examination was negative throughout except for localized tenderness in the right lower quadrant of the abdomen, and wheels of giant urticaria over the right scapula and the sacrum. The temperature was 99 F.; the pulse, 94; respiration, 22. The white blood cells were 7,000. The giant urticaria faded after two doses of 0.5 c.c. of epinephrin (1: 1,000) given hypodermically.

Appendectomy was performed through a McBurney incision, April 29. The anesthesia was ether, and was uncomplicated. The next day, April 30, when I was making rounds I found this patient lying on his right side; his lips were distinctly cyanotic, and, when he was placed on his back, the left chest was depressed and showed limited respiratory movements. Examination revealed dulness and suppressed breath sounds at the right base posteriorly. The left lung was hyperresonant, and the superficial cardiac dulness was obliterated. By percussion, the apex of the heart was 1.5 cm. inside of the left nipple line. The diagnosis of postoperative massive atelectasis was made, and this was confirmed by roentgen ray. The patient was having attacks of moderate dyspnea, and of pain radiating from the chest to the operative site, especially on full expiration, and he was having an occasional short cough, and was expectorating some thick tenacious mucopurulent sputum. During this day, the temperature and pulse mounted. The respiratory rate as recorded by the nurse was not abnormal, but it was increased during the attacks of dyspnea. The elevations of temperature persisted for six days. The patient never developed any alarming dyspnea, but the cyanosis was marked for five days. May 3, the third day after onset, bronchial breathing was well developed in the upper part of the dull area.

May 14, there were still dulness and loud bronchial breath over the right back from the angle of the scapula downward, and roentgen-ray examination, the day previous, showed opacity in this area, though the upper part of the lung had greatly cleared. On this date, the patient showed a marked hypersensitiveness to 0.5 c.c. of 1: 1,000 epinephrin. He was discharged, May 15, symptomless and feeling well, but with signs present at the right base. May 29, he returned for examination. He had gained 9 pounds in weight, and had had no symptoms. The chest was symmetrical, and the respiratory excursion was good and equal on the two sides. The percussion note was resonant throughout, and the breath sounds vesicular. By roentgen-ray examination, the lung fields were revealed to be clear, and the heart in its normal position. Fluoroscopically, parodoxic respiratory movements of the right diaphragm were present. This paresis of the right diaphragm was the only residual sign or symptom. It was further evidenced by an excursion of the right costal margin greater than the excursion on the left side. His sensitiveness to epinephrin was again tested. and he was still distinctly hypersensitive.

THE EFFECT OF LIGATION OF BRANCHES OF THE RENAL ARTERY *

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CLEVELAND

A growing tendency in operations on the kidney to regard lightly the ligation of renal arterial branches has led us to make a detailed study of the vascular changes following such a procedure.

Marion,[1] in 1922, in an article on the enlarged pyelotomy incision for the removal of large renal calculi, advocated extending the pyelotomy incision radially into the parenchyma of the kidney in the direction of the axis of the stone. In prolonging this incision, he cuts the posterior branch of the renal artery and ligates the cut ends. He states that if there is damage to the renal parenchyma following this procedure, he has seen no resulting ill effects. In a more recent article, Eisendrath [2] favors double ligation and division of the retropelvic branch of the renal artery, and states that the area supplied by the ligated vessel will receive sufficient blood from adjacent arteries which are branches of the prepelvic vessels.

A radial incision extending from the pelvis into the renal parenchyma, but confined to the lower pole, has long been advocated by Kelly [3] based on the anatomic work of Broedel. This incision, however, is calculated carefully to avoid section of the posterior branch of the renal artery, and is made in the region of the inferior pole where the finer branches of the posterior and anterior systems of arteries meet. Such an incision avoids all large arteries. It may readily be extended into the familiar longitudinal nephrotomy incision, and must be clearly distinguished from the type of radial incision into the renal parenchyma which divides the posterior branch of the renal artery (Figs. 1 and 2).

There is a well recognized class of cases in which a slowly developing hydronephrosis results from obstruction to the ureter caused by an aberrant polar artery. In this class of cases, Quinby [4] has recently

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1. Marion, G.: Le pyélotomie élargie, J. d'urol. méd. et chir. **13**:1-8 (Jan.) 1922.

2. Eisendrath, D. N.: Technique of Enlarged Pyelotomy for Renal Calculi, Surg., Gynec. & Obst. **36**:715-717 (May) 1923.

3. Kelly, H. A., and Burnam, C. F.: Diseases of the Kidneys, Ureters and Bladder, New York, D. Appleton & Co., **1**:160-164, 418-432, 444, 191.

4. Quinby, W. C.: Hydronephrosis, Tr. Am. Assoc. of G. U. Surgeons **15**:43-64, 1922; also, J. Urol. **10**:45-66 (July) 1923.

advocated a free section of the ureter followed by its reimplantation into the most advantageous portion of the pelvis, away from the vessel; and he points out that ligation of this vessel to produce a relief from obstruction is only to be advocated in those instances in which observation of its size shows it to be insignificant. On the other hand, a report by Kummer [5] reviews the recorded cases in which ligation of such

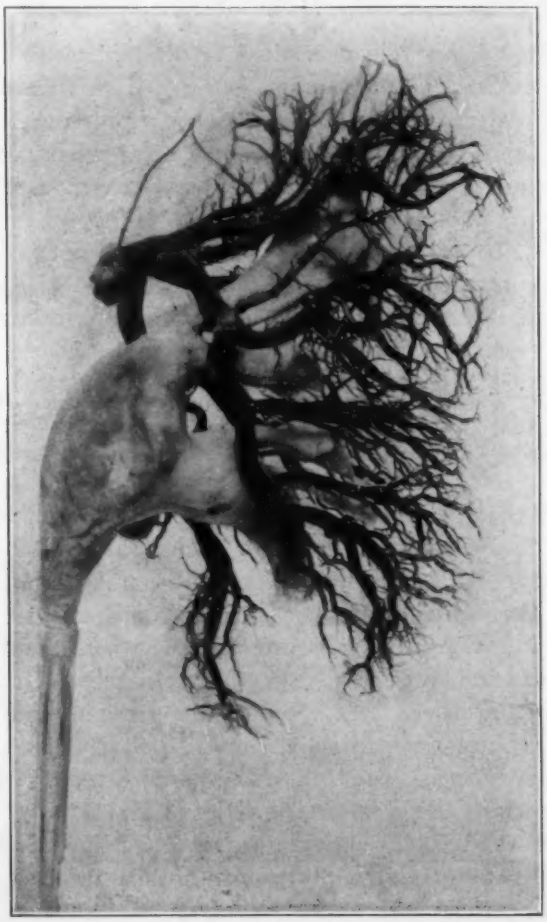

Fig. 1.—Celluloid cast of the pelvis and larger arterial branches of an adult human kidney, posterior view; constant retropelvic branch is shown with numerous interlobar branches extending from it.

aberrant obstructing vessels has been carried out. He states that, "among these fifty-six reported cases, we cannot find any complications

5. Kummer, R. H.: Résection de la bride vasculaire, traitement de choix de l'hydronéphrose due à la compression par un vaisseau anormal, J. d'urol. méd. et chir. **13**:425-432 (June) 1922.

which could lead one to believe that there is a necrosis of the kidney subsequent to the vascular section." In one of these, a case reported by Legueu, a secondary operation was done three months after the vascular section. No necrosis of the lower pole to which the aberrant vessel ran could be seen.

In contrast to this opinion are the recorded observations of pathologists who see the end-result of obstruction of renal arterial branches. Two typical examples of the results of this type of vascular occlusion are described by MacCallum.[6] In one of these, a stab wound had

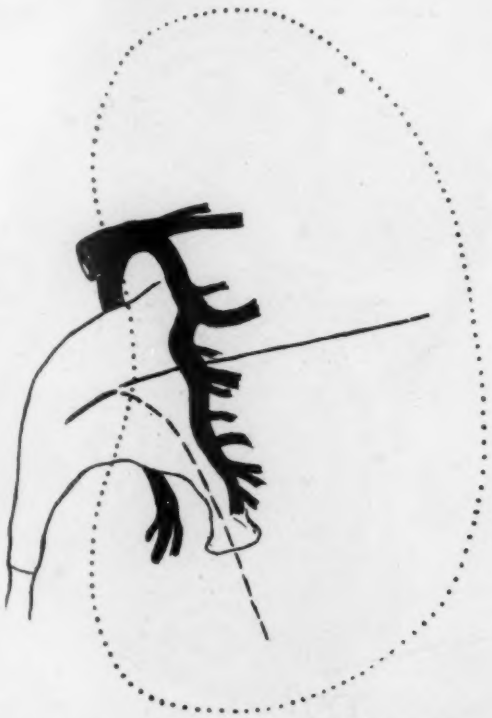

Fig. 2.—Cast shown in Figure 1, showing (dotted line) the correct and (continuous line) the incorrect direction of the extended pyelotomy incision; this shows clearly how the incorrect incision must divide the retropelvic branch, thus infarcting a considerable portion of the renal parenchyma, while the correct incision avoids injury to the larger vessels.

passed through one of the divisions of the renal artery which happened to begin its branching outside of the kidney, and a hemorrhage occurred which continued slowly for two or three days, being restrained in part by the surrounding tissues. At the end of that time, the kidney was

6. MacCallum, W. G.: A Text Book of Pathology, Ed. 1, Philadelphia, W. B. Saunders Company, 1917, pp. 27-28 and 67.

removed at operation. It was found that there was an extensive infarction occupying a portion of the kidney which should have been supplied by the injured artery. The result of longer obliteration is seen in the second case. Here there were two arteries supplying the kidney, one of which had long been occluded by a thrombus. That half of the kidney was shrunken, in sharp contrast to the other half.

Fig. 3.—Human adult kidney, posterior view; aberrant vessel capping the upper pole of the kidney injected with white celluloid; main renal vessel with red; ureter seen coursing downward past lower pole.

Careful studies of many corrosion specimens of renal arterial injections showed us that the conception of Broedel [7] is in the main correct: "the arteries are end arteries in the strictest sense, and an artery that runs anteriorly to the pelvis stays anterior until the capsule is reached. In an arterial corrosion preparation the two trees of arteries can be

7. Broedel, cited by Kelly and Burnam (Footnote 3).

separated and bent away from one another without injury to the fine branches of the cortex." It is well known that this end arterial arrangement applies also to the individual divisions of the anterior and posterior trees. If these anatomic considerations are accurate, a ligated renal vessel cannot be expected to reestablish its circulation through anastomotic branches from adjacent vessels. We have here then a conflict of opinion.

Fig. 4.—View from the hilum of the kidney shown in Figure 3; the area supplied by the aberrant vessel has been easily removed without breaking any connecting vessels; the superior calyx has been exposed by this procedure.

In this work, an attempt has been made to determine by arterial injections whether or not there is sufficient anastomosis present between the branches of the renal artery to allow promiscuous ligation of its larger branches. Three types of kidneys were studied: (*a*) normal kidneys of man and dog; (*b*) human kidneys, obtained at necropsy, which were partly supplied by an aberrant polar artery; (*c*) dogs' kidneys in which an experimental infarct had been produced by ligating the anterior division of the renal artery.

EXPERIMENTAL PROCEDURE

In order to study the results of ligation of branches of the renal artery, we did our experimental work on dogs because in this animal the main renal artery usually divides just outside the kidney hilum into an anterior and posterior branch of about equal size (Fig. 6). Furthermore, although the single pyramid of the dog's kidney allows a somewhat simpler arterial arrangement, nevertheless, the distribution of these vessels is very similar to that found in the human kidney. Especially is this so in the arrangement of the finer branches which are as truly end arteries as those of man.

Desiring to bring about experimental conditions which could give every opportunity for regeneration of the damaged renal segment if such regeneration was possible, we decided to ligate the anterior division of the renal artery on each side of the body, thus producing a real reduction of renal substance with consequent body demand for renal tissue.

All operations were performed under ether anesthesia and with aseptic technic. A midline incision was made from the zyphoid to below the midabdominal region Each kidney was exposed, and the anterior branch of each renal artery isolated and freed backward to its junction with the posterior branch. It was here doubly ligated and cut. After intervals of forty-eight hours, seventy-two hours, one week, two weeks, three weeks, five weeks and seven weeks, these dogs were killed with ether and the kidneys carefully removed. The gross and minute vascular structures were studied by means of the celluloid corrosion method described by Huber.[8] A solution of celluloid dissolved in acetone was injected into the renal artery under sufficient pressure and of sufficient dilution to obtain casts of the arterial tree down to the first and sometimes as far as the second capillary bed. Corrosion was effected by means of concentrated hydrochloric acid.

ANATOMIC FINDINGS

Normally, on reaching the kidney hilum, the renal artery in the human being divides into two main branches, an anterior and a posterior. The anterior or prepelvic division usually supplies approximately the anterior three-quarters of the renal parenchyma, and the posterior or retropelvic branch the remaining one-quarter. However, this is not constant, so that the two divisions may be equally large or the posterior even the larger. In dogs, the two divisions are usually approximately equal (Fig. 6).

After reaching the hilum, the anterior division of the renal artery soon divides into a number of smaller branches, known as the interlobar

8. Huber, G. C.: The Arteriolae Rectae of the Mammalian Kidney, Am. J. Anat. **6**:391-406, 1906-1907.

arteries, which radiate from the hilum in a fanlike manner. The posterior or retropelvic division runs downward and outward over the posterior aspect of the intrarenal portion of the pelvis, terminating over the posterior inferior calyx (Fig. 1). In its course, it gives off the radiating interlobar arteries to the posterior portion of the kidney.

Fig. 5.—Anterior surface view of normal kidney of dog; corrosion specimen, arterial injection. Myriads of glomeruli can be seen as minute spheres studding the surface of the kidney. A perforating branch to the perirenal tissues makes its exit in the region of the upper pole; it can be seen anastomosing with a small branch from the main renal artery. The blood supply to the ureter can also be seen.

While a great deal of variation is found in all the other larger renal arteries, the retropelvic branch is remarkably constant in its distribution.

Fig. 6.—Same kidney as shown in Figure 5; the anterior and posterior trees of vessels have been separated showing the approximately equal areas supplied by the anterior and posterior divisions of the renal artery. Separation has been effected along the natural plane of cleavage. A perirenal anastomotic vessel connects the two divisions. The pelvis has also been injected and is seen in situ.

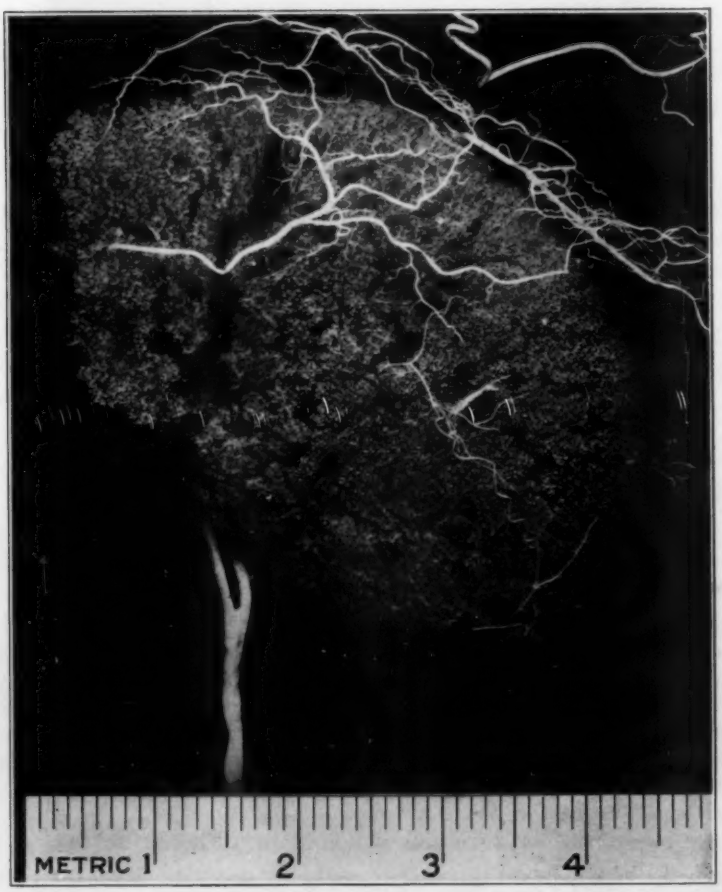

Fig. 7.—Segment of an injected dog's kidney; a perforating renal artery richly anastomosing with perirenal vessels and with smaller perforating renal arteries.

When any branch from the hilum into the kidney substance is followed, a typical general arrangement is seen. The interlobar vessels course in an arching manner toward the cortex, passing between the pyramids of the medulla, then over the bases of the pyramids where the arc is increased to form the arcuate arteries. From the cortical aspect of these arteries, branches arise which pass directly outward into the cortex, and are known as intralobular or cortical arteries. The arching branch itself also terminates in the cortex by division into multiple smaller branches.

The intralobular arteries break up into numerous fine vessels which supply the glomeruli. Occasionally, one of these branches may directly break up into a capillary bed without having first traversed a glomerulus. This, however, is rare, and with this exception every branch runs into its glomerulus. The intralobular arteries, on reaching the subcapsular region, may break up in three ways:

1. They may become terminal branches, which form a capillary cortical plexus.

2. More frequently, these vessels terminate as afferent vessels to glomeruli.

3. In some cases, the caliber remains large, and the vessel may perforate the capsule to supply the perirenal tissues (Figs. 5 and 7). Such vessels always bear glomeruli in their course through the parenchyma.

Whether any artery within the kidney substance may form a capillary bed without having first traversed a glomerulus is a point disputed in the literature. Our findings, as stated above, show that a very small percentage do so distribute themselves.

The renal capillary bed is therefore derived from:

1. Vessels leaving the glomeruli (efferent vessels). These spread themselves in a plexus round the tubules of the cortex. Those from the glomeruli lying in the corticomedullary zone pass to the tubules of the medulla in a series of straight bundles (arteriolae rectae), or these capillaries may anastomose in the lower cortical zone to form small vessels which course downward toward the medulla, and here break up into another capillary bed.

2. A more rare type of capillary bed is that derived from the intralobular arteries, directly, without having traversed a glomerulus. Such capillaries distribute themselves over the surface of the convoluted tubules of the cortex probably as nutrient vessels.

The origin of the straight vessels which stream in bundles from the lower third of the cortex down into the medulla has been the occasion of a difference of opinion among observers of the renal capillary system.

Many believe that these arise as direct branches from the concave aspect of the large arterial trunks, and call such branches "arteriolae rectae verae." We believe that this conception is erroneous and has arisen through failure to use methods which employ binocular vision. With

Fig. 8.—Anterior view of injected specimen of a dog's kidney forty-eight hours after severance of the anterior division of the renal artery; specimen shows only the faintest tracery of arteries in the infarcted half of the kidney. No glomeruli are present on these vessels. The practically complete absence of arteries in the infarcted half gives the specimen an appearance of only a separated posterior half of a dog's kidney (comparison should be made with Figures 6 and 17). A ureteral vessel can be seen at the hilum of the kidney just above the posterior division of the renal artery; this ureteral artery anastomoses with the vessels of the infarcted side.

binocular vision, it is easy to see that the straight vessels, having arisen as afferent glomerular vessels, pass close to and around the large arterial trunks in the corticomedullary zone. If seen in thin sections, they may readily appear to arise from these vessels. Extremely rarely, branches may arise as the terminal ramifications of a glomerular bearing artery which has dipped into the small area of cortex in the interpyramidal zone. These branches run down among the tubules of the medulla as straight vessels.

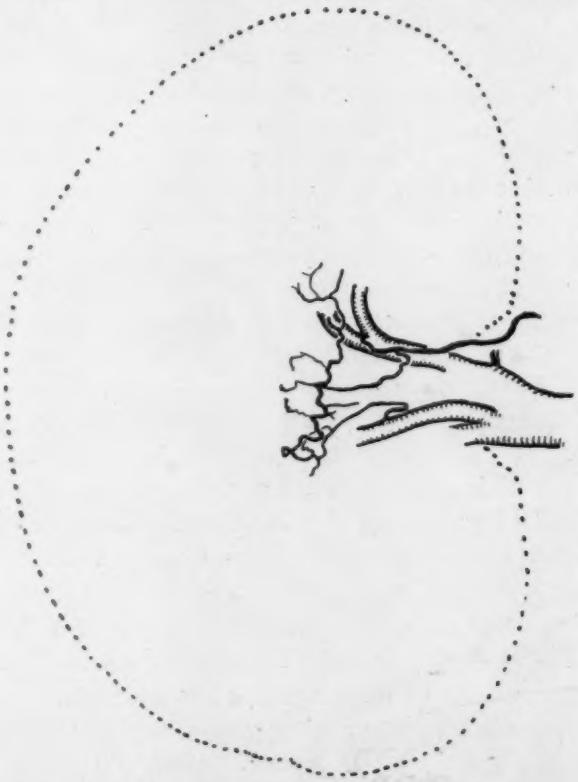

Fig. 9.—Outline drawing of photograph shown in Figure 8 to indicate more clearly the small vessels of the infarcted portion of the kidney; the ureteral artery mentioned is outlined by a heavier line just above the large vessels.

A point of great importance to the subject under discussion is the demonstrable absence of any vessels of capillary size in the kidney parenchyma which could connect the anterior with the posterior renal artery or even connect one intralobular vessel with its neighbor. The only vessels visible in normal specimens through which such a connection is possible are small branches from the large renal arteries which furnish nutrition to the pelvic and ureteral wall and those interlobar vessels which perforate the kidney capsule and take their subsequent course in the perirenal fat (Fig. 7).

EXPERIMENTAL RESULTS

In human kidneys, removed at necropsy and injected with a celluloid injection mass fine enough to fill the blood vessels down to the efferent system of capillaries, if previous to injection one of the branches of the renal artery was ligated, the corrosion specimen showed a complete absence of any of the injection mass in the region supplied by the ligated vessel. This region appeared as a punched out hole which was surrounded by the characteristic dense network of renal vessels and capillaries. Evidently, no anastomotic channels were present through which the injection mass could flow from the unligated vessels into the blocked region. Similar results were obtained in kidneys which were partly supplied by a polar artery. When only the main renal artery and not the polar artery was injected, the portion of the kidney supplied by the aberrant renal vessel showed a complete absence of injected vessels, and no anastomotic branches from adjacent vessels were revealed. When the main renal artery was injected with a red celluloid solution and the polar artery with a white solution of celluloid, the corrosion specimens showed these two divisions of the circulation to be absolutely independent of each other, and no anastomotic branches whatsoever could be found. The two groups of vessels could easily be separated from each other without breaking any connecting branches (Figs. 3 and 4).

In dog kidneys in which infarcts had been brought about by ligation of the anterior division of the renal artery, somewhat different results were obtained. Here, small but definite anastomotic channels were found to have opened between the posterior and anterior trees of arteries. These channels were of three main types.

1. *Pelvic and Ureteral Anastomoses.*—In the pelvic anastomoses, the pelvis of the kidney is supplied by branches from both the anterior and the posterior trees of renal arteries, and these pelvic vessels anastomose very freely with each other within the pelvic wall. In the ureteral anastomoses, the ureter is also supplied by branches from both the anterior and posterior trees which anastomose freely with each other within the ureteral wall (Figs. 5 and 13).

2. *Perirenal or Capsular Anastomoses.*—The perirenal vessels may arise as perforating arteries through the parenchyma of either the anterior or the posterior surfaces of the kidney (Figs. 5, 7 and 10), or at the hilum of the kidney from either the anterior or posterior branch of the renal artery (Figs. 10 and 11), or extrarenally (Fig. 14). These perirenal vessels anastomose very freely outside the true capsule of the kidney in the perirenal fat.

3. *Small Intrarenal Anastomotic Vessels.*—These run directly between the main branches of the posterior and the anterior divisions

of the renal artery (Figs. 17, 21 and 23). These usually run in the region of the corticomedullary junction, but may also pass through the medulla and even through the cortex.

The size of these anastomotic channels and the number and size of the arteries which appeared in the infarcted portion of the corrosion

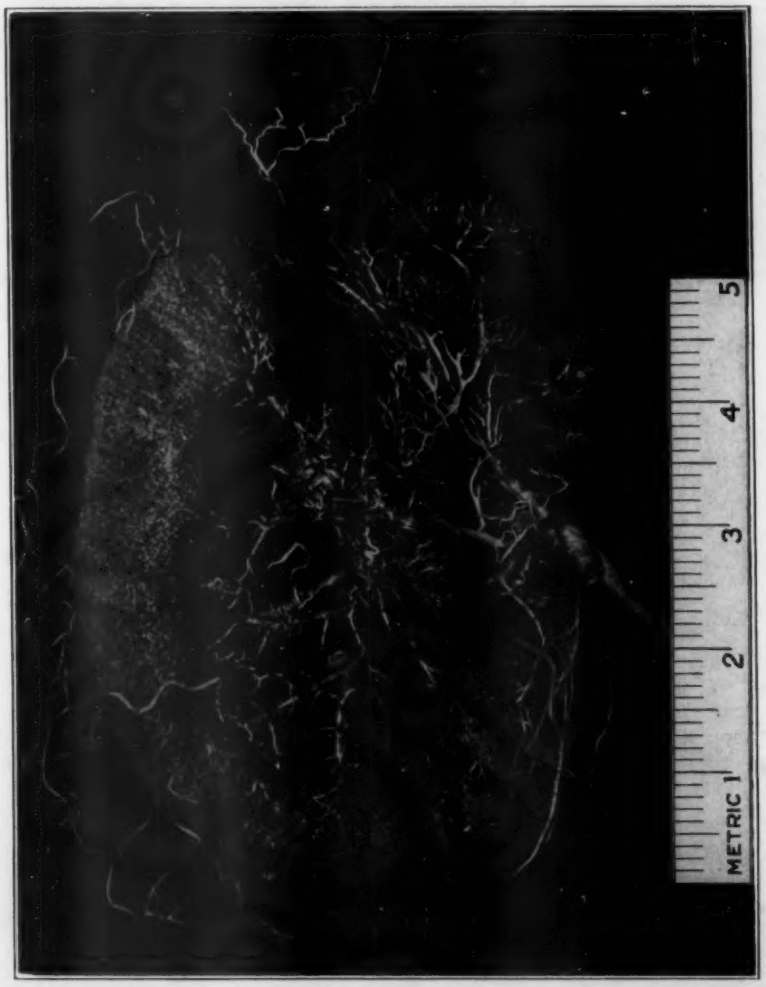

Fig. 10.—Anterior view of injected specimen of a dog's kidney seventy-two hours after severance of the anterior division of the renal artery; there has been a marked increase over the forty-eight hour specimen in the appearance of vessels in the infarcted area. However, no glomeruli are present. Perirenal anastomosis can be seen. The posterior portion of the kidney shows the usual rich supply of glomeruli. The upper pole of the posterior portion has been removed in order to show more clearly the type of vessels present in the anterior infarcted portion.

specimens, up to a cretain point, varied in direct proportion to the length of time which had elapsed since the production of the infarct. Thus, in the forty-eight hour specimens (Fig. 8), the anastomotic channels were very fine, and the arteries which appeared in the infarcted portion of the kidney were very small and scant, and bore no glomeruli.

Fig. 11.—Cross section of injected specimen of a dog's kidney seventy-two hours after severance of anterior division of renal artery showing a fine subcortical intrarenal vessel arching across from a large vessel of the posterior tree to a large vessel of the anterior infarcted portion; some perforating arteries can be seen at the top of the picture dipping into the infarcted area.

In the seventy-two hour specimens (Figs. 10 and 11), the vessels in the infarcted area showed a marked increase in size and number over

the forty-eight hour specimens, but still no glomeruli were present. In a week, the anastomotic channels had become somewhat larger, and the vessels in the infarcted area were definitely larger and more numerous (Fig. 13). They subdivided into numerous fine branches which bore no glomeruli. In two weeks, the vessels of the infarcted area were still larger, and some glomeruli were present on the finer branches (Figs. 14 and 16). At three weeks, there was still a further increase in glomeruli. However, the entire size of the anterior infarcted area of the vessels was much less than that of the unligated posterior tree (Fig. 19). In the five week specimens, the greatest number of glomeruli had

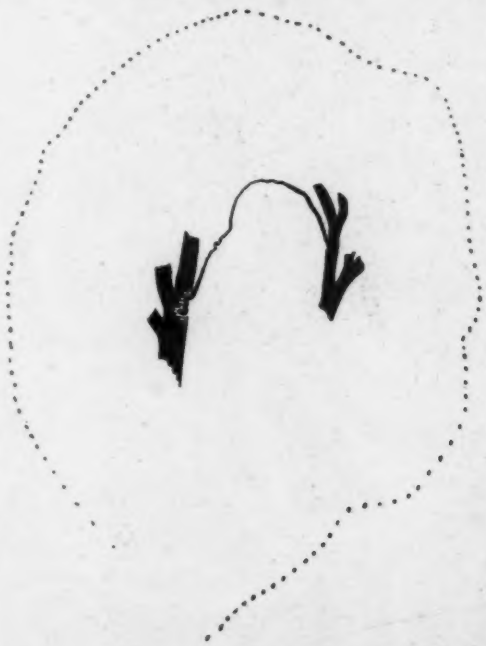

Fig. 12.—Outline drawing of specimen shown in Figure 11 tracing the course of the subcortical intrarenal vessel described.

reappeared in the infarcted area, but even then their number was exceedingly small as compared with the vast number to be found in the uninfarcted portion or in a normal kidney (Figs. 20 and 21). The next specimen injected was a kidney in which an infarct had been produced seven weeks previously The corrosion specimen here showed the infarcted region to be markedly shrunken. The main vessels were of fairly good size and well filled. They divided into numerous smaller branches, but careful examination of the specimen showed that no glomeruli were present although the posterior uninfarcted portion showed the usual rich supply of glomeruli (Fig. 23).

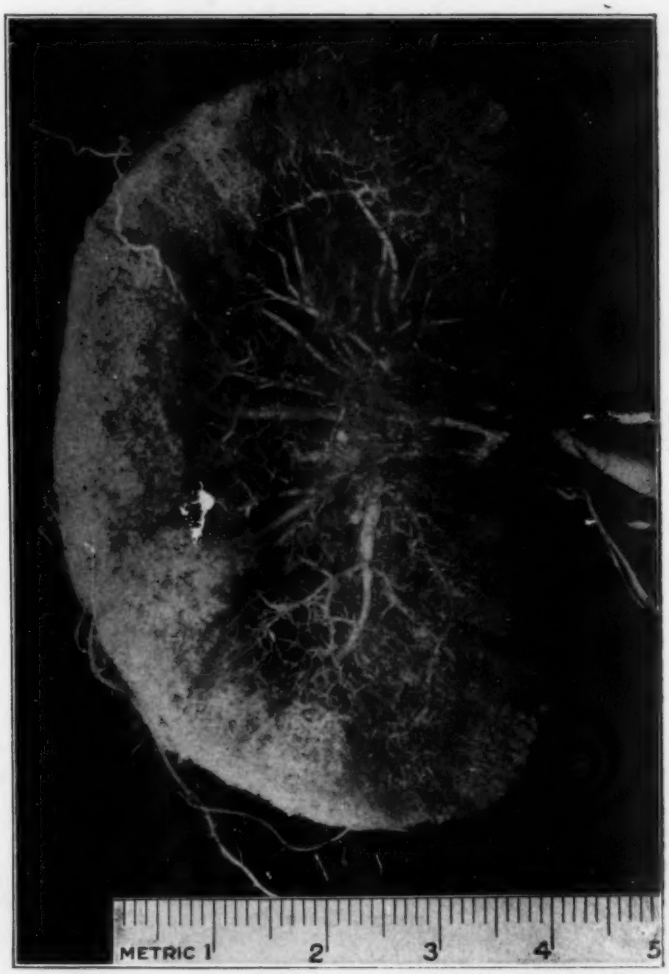

Fig. 13.—Anterior view of injected specimen of a dog's kidney one week after severance of anterior division of renal artery. The stump of this vessel is evident on the side of the renal artery. The vascular channels in the infarcted area are reappearing but are barren of glomeruli. Note should be made of the richness of glomeruli in the uninfarcted area. A perirenal artery is seen dipping into the infarcted area and anastomosing with the arteries of this area. Ureteral anastomoses are also evident.

PROTOCOLS

EXPERIMENT 1.—A forty-eight hour infarct (Fig. 8) in a mongrel, female collie, weighing 19 kg., was operated on, May 31, 1923, under ether anesthesia; a transabdominal exposure was made. The artery supplying the anterior half of each kidney was cut between ligatures at the kidney hilum.

Necropsy was done, June 2, forty-eight hours after operation. The general examination was negative. The anterior surfaces of both kidneys were pulpy and the overlying capsule edematous. The infarcted area was pale and surrounded by a narrow reddish zone.

In the injected specimens, the posterior tree of the renal vessels of the left kidney was of normal size and well filled. The usual rich supply of glomeruli was present. The anterior infarcted half of the kidney was practically entirely missing. Only the faintest tracing of arteries could be seen in this area, and these were very small. Relatively few finer branches arose from the main vessel, but no glomeruli were seen. Anastomoses between the posterior tree of vessels and the infarcted area took place through fine pelvic and ureteral branches, and through a small intrarenal artery which arose from one of the primary branches of the posterior division of the renal artery and ran anteriorly through the medullary region of the kidney to join one of the larger vessels of the infarcted area.

The right kidney had the same general appearance as the left, i. e., a normal posterior half of the kidney and a very poorly supplied anterior infarcted portion which contained no glomeruli. Anastomoses between the posterior half of the kidney and the infarcted portion took place only through pelvic and ureteral branches.

EXPERIMENT 2.—A seventy-two hour infarct (Figs. 10 and 11), in a short-haired female mongrel, weighing 16 kg., was operated on, June 28, 1923; the artery to the anterior half of each kidney was doubly ligated and severed.

July 1 (seventy-two hours after operation), necropsy was done. The general examination was negative. There was definite softening of the anterior portions of both kidneys. This softened area was paler than the remainder of the kidney tissue, and the overlying capsule was dull and slightly edematous.

In the injected specimens, the posterior portions showed the usual rich supply of glomeruli. The anterior infarcted portions showed a remarkable increase in the size and the number of the arteries over the forty-eight hour specimens. However, no glomeruli were present. Anastomoses between the two portions of the kidney took place by means of perforating arteries and pelvic and ureteral anastomoses. All these anastomotic channels were very fine in caliber.

EXPERIMENT 3.—A one week infarct (Fig. 10), in a male collie, weighing 21 kg., was operated on, May 16, 1923; the artery to the anterior half of each kidney was cut between metal clips.

May 23, one week after operation, necropsy was done. The general examination was negative. The anterior surfaces of both kidneys were paler than normal. The infarcted areas appeared slightly depressed.

In the injected specimens, the posterior portion of the right kidney showed the normal rich supply of glomeruli. The large vessels of the infarcted portion were well filled. They were smaller than those of the posterior tree and subdivided into numerous very small vessels, but these bore no glomeruli. Only one very small tuft of glomeruli was present, and this was supplied by

Fig. 14.—Anterior view of injected specimen of dog's kidney two weeks after severance of anterior division of renal artery; attention is called to the perirenal artery coursing along the convex border of the kidney, and sending perforating branches into the infarcted area. This large perirenal vessel evidently arose extrarenally. A small perirenal artery is seen arising at the hilum and swinging around to the anterior surface of the kidney.

a very fine intrarenal artery which ran through the cortex from the posterior tree to the infarcted area, and finally anastomosed with one of the larger branches. Other anastomoses took place through perforating arteries and pelvic and ureteral arteries. These anastomotic channels were larger than those of the seventy-two hour specimen, but were still very fine vessels.

The left kidney showed the same general appearance. No glomeruli were present in the infarcted area. Anastomoses between the posterior tree and the infarcted area took place through perforating branches, pelvic branches, and through an intrarenal vessel which ran subcortically from a main posterior branch to a larger anterior artery.

Fig. 15.—Outline drawing of kidney shown in Figure 14 pointing out more clearly where the perforating vessels shown in the photograph dip into the infarcted area.

EXPERIMENT 4.—A two week infarct (Figs. 14, 16 and 17), in a female mongrel, weighing 13.5 kg., was operated on, May 9, 1923; the artery to the anterior half of each kidney was doubly ligated and severed.

May 23, two weeks after operation, necropsy was done. The general examination was negative. The anterior surfaces of both kidneys were flattened. The infarcted areas were paler and firmer than the normal portions of the kidneys.

In the injected specimens, the posterior portion of the right kidney showed the presence of the usual myriads of glomeruli, while the anterior portion was very poor in glomeruli, but there was definitely a small number of them present. Two perirenal anastomotic channels were present. The larger of

these passed from the uninfarcted side around the convex border of the kidney and dipped into the infarcted portion of the kidney to anastomose with a thick tuft of vessels on which the glomeruli were more numerous than elsewhere in the infarct. The smaller of the perirenal vessels passed from the posterior portion of the kidney over the superior pole to the infarcted region where it anastomosed with arteries of this area. Anastomoses also took place through some pelvic branches, but no other anastomotic channels were present.

The general appearance of the specimen of the left kidney was the same as the right. A small number of glomeruli were present in the infarcted area. A large perirenal artery was present which seemed to arise extrarenally. This vessel coursed along the convex border of the kidney and sent a large branch over the anterior surface of the kidney which dipped into the infarcted area near the hilum and anastomosed with the vessels of this region. The continuation of the perirenal artery spread itself over the upper portion of the anterior surface of the kidney, and in three places dipped into the infarcted area to anastomose with the arteries of this region. Another small perirenal vessel arose from the main renal artery near the hilum and ran up over the anterior surface of the kidney, to aid in the supply of the infarct. Pelvic anastomoses also were present. A small intrarenal artery arose from the middle primary branch of the posterior division of the renal artery and ran anteriorly through the region of the corticomedullary junction to anastomose with one of the larger vessels of the infarcted area.

EXPERIMENT 5.—A three week infarct (Fig. 19), in a female mongrel, weighing 16.9 kg., was operated on, March 19, 1923; the artery to the anterior half of each kidney was cut between metal clips.

April 9, three weeks after operation, necropsy was done. The general examination was negative. Both kidneys showed a contracted infarcted anterior region with definite hypertrophy of the posterior portions of the kidney.

The posterior portion of the left kidney was well injected and normally rich in glomeruli. The anterior infarcted portion showed a definite increase in glomeruli over the two week specimen, but the number of glomeruli was still very far below that seen in the uninfarcted portion of the kidney. Two small perirenal vessels formed connecting channels between the anterior and the posterior sets of vessels. Five small pelvic vessels arose from the various primary branches of the posterior division of the renal artery and anastomosed with similar branches of the anterior division. There was a fine subcortical artery which arose from the middle primary branch of the posterior division, and ran straight across to the primary branch of the anterior division. A fairly large ureteral branch arose from the most inferior primary branch of the anterior and posterior sets of vessels, and these two ureteral vessels anastomosed freely in the wall of the ureter.

The right kidney showed essentially the same picture except that two subcortical anastomotic branches were present instead of one.

EXPERIMENT 6.—A five week infarct (Figs. 20 and 21), in a mongrel, male collie, weighing 13 kg., was operated on, April 27, 1923; the artery to the anterior half of each kidney was cut between metal clips.

June 1, five weeks after operation, necropsy was done. The general examination was negative. The anterior surfaces of both kidneys showed markedly flattened and contracted infarcted areas. There had been definite hypertrophy of the posterior portions of the kidneys.

In the injected specimens, the posterior portion of the right kidney showed the usual rich bed of glomeruli which overlapped the convex border of the

kidney and extended somewhat into the anterior half. The infarcted portion of the kidney showed the main vessels well filled and of good size. They were only slightly smaller than the main vessels of the posterior tree and they subdivided into numerous fine arteries which bore glomeruli. The number of glomeruli showed a definite increase over the number noted in the three week specimen, but the richness of the supply in no way compared with that of

Fig. 16.—View of the inner aspect of separated infarcted portion of kidney shown in Figure 14; very few scattered glomeruli are seen as compared with the uninfarcted side.

the normal side. The anastomotic pelvic arteries between the anterior and posterior trees were fairly large in this specimen. One small perirenal anastomotic artery was present. An intrarenal anastomotic artery arose from one of the lower primary branches of the posterior tree and ran anteriorly

through the medullary region to anastomose with two of the primary branches of the infarcted area. Another intrarenal artery arose at about the middle of a primary branch of the posterior tree and followed a tortuous course anteriorly to join one of the primary branches of the infarcted portion near its termination in the cortex. Still a third intrarenal artery was present which came from one of the primary branches of the posterior tree in the region of the corticomedullary junction and ran toward the region of the hilum of the kidney where it turned and ascended to join a large artery of the anterior tree at about its middle.

The left kidney had the same general appearance. The number of glomeruli in the infarcted portion appeared to be about the same as on the right side. Anastomoses between the posterior and anterior trees took place through pelvic and ureteral arteries and two intrarenal anastomotic arteries. No perirenal anastomoses were present.

EXPERIMENT 7.—A seven week infarct (Fig. 23) in a male Great Dane, weighing 21.6 kg., was operated on, May 11, 1923; the artery to the anterior half of each kidney was cut between metal clips.

June 29, seven weeks after operation necropsy was done. The general examination was negative. The kidneys showed a deeply depressed, firm area on the anterior surface. The infarcted area was markedly contracted and there had been hypertrophy of the posterior portion, so that it extended well onto the anterior surface of the kidney.

In the injected specimens, the posterior portion of the left kidney showed the usual supply of glomeruli. The anterior infarcted portion was small and contracted. Its main vessels were well filled and fairly large, but not as large as those of the posterior tree. These main branches of the anterior tree subdivided into numerous finer branches, but careful examination revealed no glomeruli present. A large perirenal vessel coursed around the convex border of the kidney uniting the anterior and posterior sets of vessels. A fairly large intrarenal subcortical vessel also formed a connecting link between these two sets of vessels. Anastomoses by means of pelvic and ureteric arteries were also present, and these were of fairly good size.

COMMENT

It was pointed out, long ago, that certain organs possessed an arterial circulation in which single branches did not anastomose with one another through arterioles or larger vessels, but only after reaching the capillary bed. Cohnheim [9] referred to such a circulation as "terminal," and to such arteries as "end arteries." He believed that such an arrangement occurred in brain, retina, lung, spleen and kidney, and pointed out that this arterial arrangement accounted for the severe damage seen in these organs on occlusion of a part of their arterial supply.

In the kidney, the resulting damage from arterial occlusion has been frequently studied. Excellent pictures and descriptions of gross and microscopic appearance of the changes in the areas damaged by such occlusion abound in the literature. The area of distribution of the ligated vessel is first marked by bluish discoloration over the kidnev

9. Cohnheim, cited by Litten (Footnote 20).

surface.[10] This is succeeded by faint pallor in the central area of the infarct, and slightly reddened edges. The infarcted area, during the first few days, is somewhat raised and has an edematous, watery appearance. Microscopic study of the central portion reveals marked necrosis of all elements. Surrounding this is a hemorrhagic area showing congestion of all vessels and some extravasation. Between this and healthy tissue is a narrow, pale boundary zone in which some of the tubule cells have undergone coagulation necrosis, while in others the cytoplasm stains faintly.

Foa [11] describes and pictures the gross and microscopic changes at varying intervals after the ligation of branches of the renal artery. Slowly, the area of maximal injury shows more and more connective tissue. Through the shrinkage of the latter, the infarct grows smaller, and the end defect is finally a more or less broad scar which, through traction on the capsule, forms a dimple shaped depression. The changing picture is well described by MacNider.[12] On section, the cortex is found to be greatly decreased. There is extensive degeneration of the epithelium of the tubules, and in places the epithelium is entirely wanting. The connective tissue overgrowth is diffuse and abundant, and in general has replaced degenerated tubules. Glomeruli vary in size depending on the stage of fibrosis. Bowman's capsule is thickened. When the fibrosis is complete in the glomeruli, they appear as small fibrous masses with beginning hyaline changes. The smaller vessels show distinct obliterative changes affecting principally the endothelium and leading to an endarteritis obliterans. There is a marked line of demarcation between the tissues supplied by the infarcted vessels and the neighboring tissues adequately supplied with blood. Where these two portions of the kidney merge into one another, the epithelium shows advanced cloudy swelling and necrosis, and the connective tissue hyperplasia, which is commencing, is in the cellular stage. This fibrous overgrowth and associated degeneration is progressive. In some areas, fibrosis becomes complete and all evidence of kidney tissue disappears. In other areas, the renal epithelium is partially preserved.

A feeble attempt at regeneration of the renal epithelium, after infarction, has been described by practically all observers. This is in a

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10. Harrington, S. W.: Effect on Kidneys of Various Surgical Procedures on Blood Supply, Capsule and Ureters, Arch. Surg. **2**:547-592 (May) 1921.

11. Foa, P.: Ueber Niereninfarcte, Experimentaluntersuchung, Beitr. z. path. Anat. u. z. allg. Path. **5**:275-289, 1889.

12. MacNider, William: The Pathological Changes Which Develop in the Kidney, as a Result of Occlusion, by Ligature of One Branch of the Renal Artery, J. M. Res. **17**:91-94 (Feb.) 1910; The Pathological Changes Which Develop in the Kidney, as a Result of Occlusion, by Ligature of One Branch of the Renal Artery, Part II, Conclusion, J. M. Res. **19**:425-454 (April) 1911.

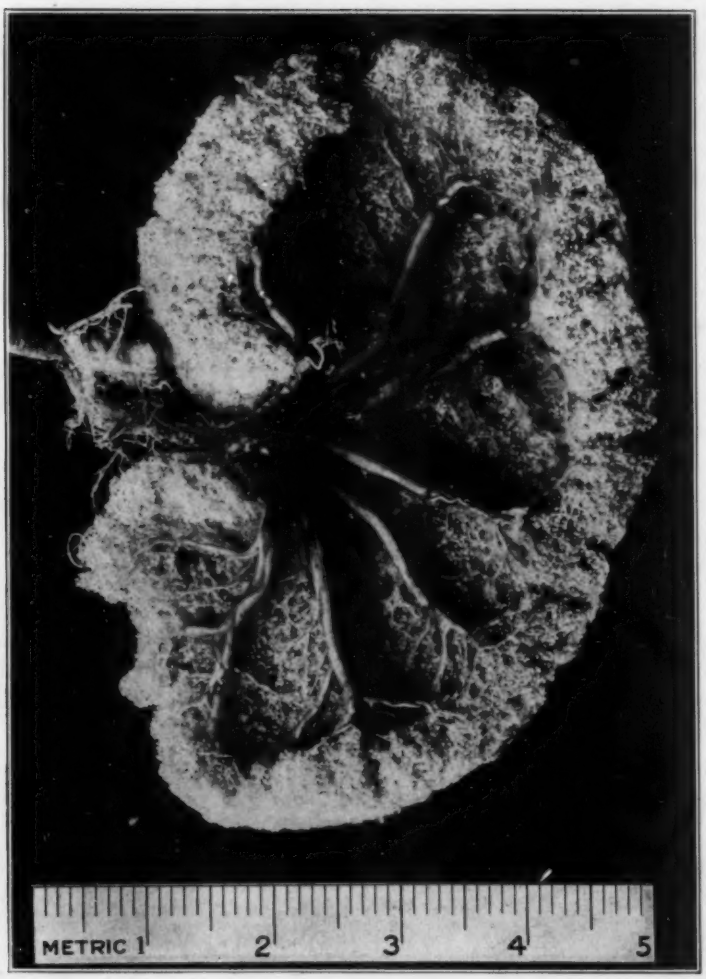

Fig. 17.—View of inner aspect of uninfarcted portion of kidney shown in Figure 14; a fine subcortical intrarenal artery which connected the anterior and posterior trees of vessels has been broken in the separation of these two portions of the kidney. This vessel can be seen arising from one of the main arteries of the posterior tree. Comparison should be made with Figure 16.

large measure confined to the marginal zone contiguous to the healthy tissue. Even here, true regeneration of tubules and glomeruli is emphatically denied by Thorel,[13] and the described regeneration is evidently a weak attempt toward new production of epithelium in areas in which it has been lost through infarction. In transplanted kidney tubules, especially if the transplant be carried to a region where the capillary supply is very rich, Loeb[14] has described a like tendency toward epithelial proliferation. Mitotic proliferation, intense at first, begins to decrease markedly after the seventh day. A like proliferative stimulus is shown by Carrel and Burrows[15] to occur in kidney tissue

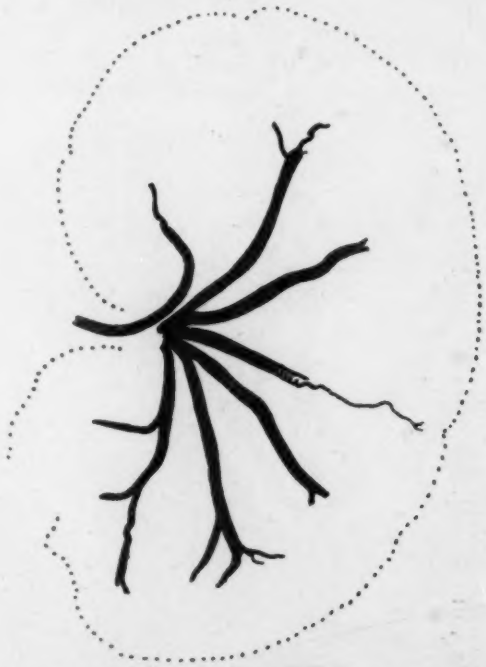

Fig. 18.—Outline drawing of the photograph shown in Figure 17 to localize the small subcortical intrarenal anastomotic branch described.

placed in properly prepared plasmatic mediums. Hence, it is not surprising that mitotic figures are common in the regenerating renal epithelium at the margins of infarcts where there must be a certain stimulus received from the rich capillary supply in the neighboring undisturbed regions.

13. Thorel, C.: Ueber typische und Pseudoregeneration bei Niereninfarcten, Virchows Arch. f. path. Anat. **146**:297-330 (Nov. 4) 1896.

14. Loeb, L.: Further Investigations on Auto and Homoioplastic Transplantation of Kidney Tissue, J. M. Res. **32**:229-246 (Nov.) 1917.

15. Carrel, A., and Burrows, M. T.: Cultivation of Adult Tissues and Organs Outside of the Body, J. A. M. A. **55**:1379-1381 (Oct. 15) 1910.

Fig. 19.—Injected specimen of the infarcted half of a dog's kidney three weeks after severance of anterior division of renal artery; there is an increase in glomeruli over the two week specimen (comparison should be made with Figure 16). The injected ureter has been left in place, but the pelvis has been removed. The posterior portion of the kidney appeared normal, similar to that seen in Figures 6 and 17.

Many observers, Brodie [16] Cushny [17] and Overbeck,[18] have claimed that temporary occlusion of the renal artery resulted in varying but considerable periods of anuria even after the circulation has been reestablished. More recently, Marshall and Crane [19] have demonstrated that this is not so. However, Litten [20] has shown that longer periods of temporary anemia do cause death of some of the renal parenchyma, thus demonstrating the sensitiveness of the epithelium to anemia. He ligated individual branches of the renal artery in rabbits, and at the end of

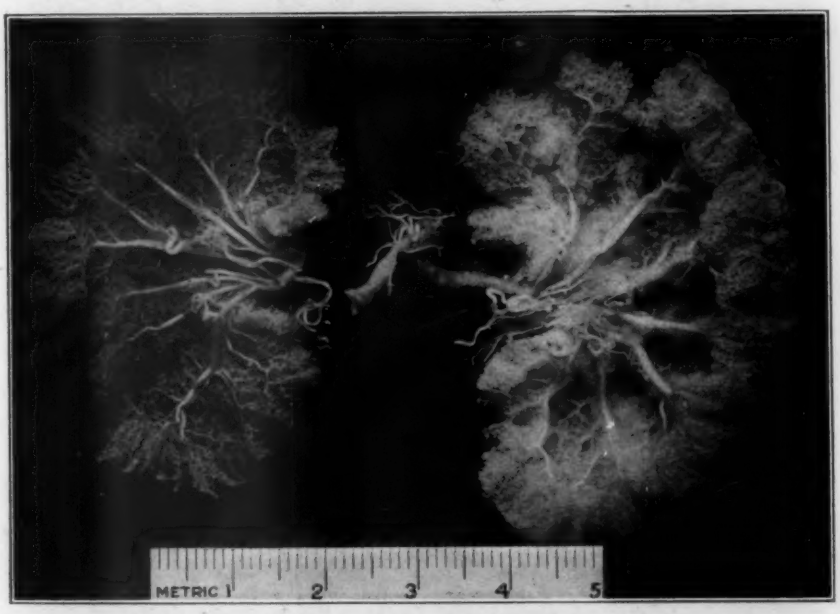

Fig. 20.—Injected specimen of a dog's kidney five weeks after severance of anterior division of renal artery; the infarcted area has been separated from the posterior portion of the kidney. Both sides are viewed from the inner aspect. There has been a remarkable reappearance of glomeruli and finer branches in the infarcted side, but the richness of the supply in no way compares with that of the normal side. The fairly large anastomotic vessels seen in the pelvic region were divided when the two halves of the kidney were separated.

16. Brodie, T. G.: Croonian Lecture: A New Conception of Glomerular Function, Proc. Roy. Soc. **87**:571-592, 1914.

17. Cushny, A. R.: The Secretion of Urine, New York, Longmans, Green & Co., 1919, p. 198.

18. Overbeck: Sitzungsb. d. Math. Natur. Classe **47**:11-189, 1863.

19. Marshall, E. K., and Crane, M. M.: The Influence of Temporary Closure of the Renal Artery on the Amount and Composition of Urine, Am. J. Physiol. **64**:387-403 (April) 1923.

20. Litten, M.: Untersuchungen über den haemorrhagischen Infarct und über die Einwirkung arterieller Anaemie auf das lebende Gewebe, Ztschr. f. klin. Med. **1**:131-227, 1880.

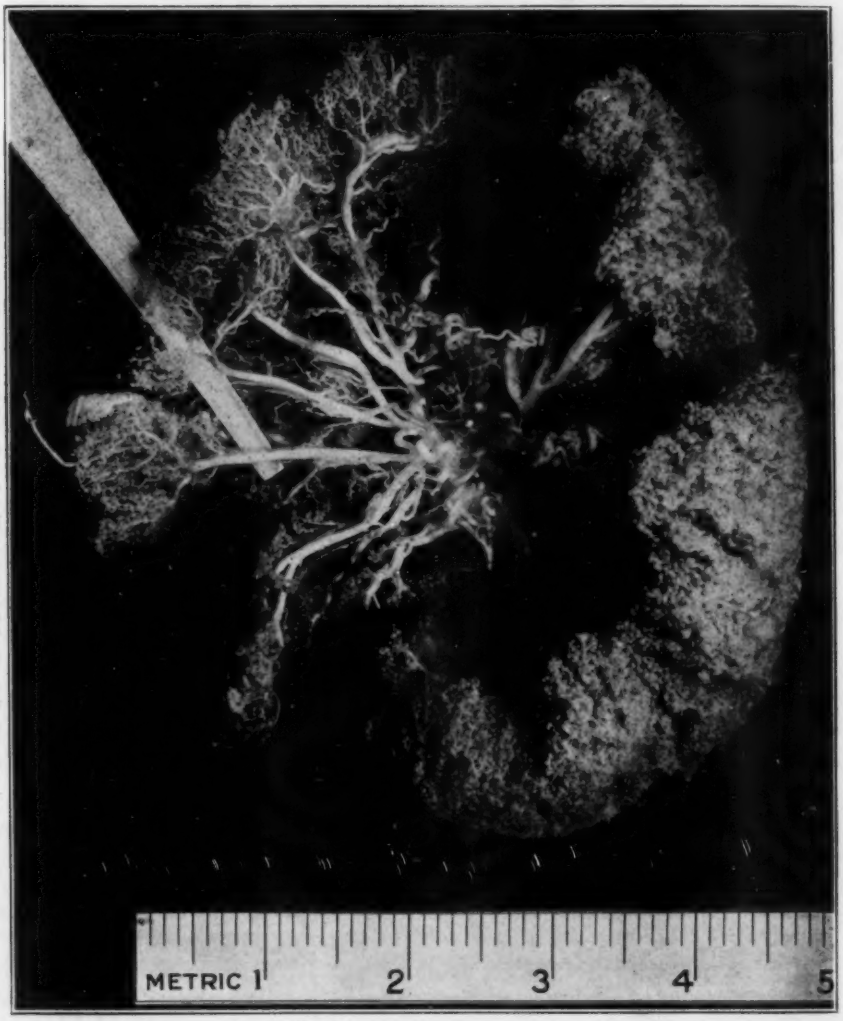

Fig. 21.—Injected specimen of a dog's kidney five weeks after severance of the anterior division of the renal artery; the anterior and posterior trees have been partially separated in order to show an intrarenal anastomotic branch running directly across from one of the main vessels of the posterior tree to the main vessels of the anterior tree (Fig. 22).

from one and one-half to two hours opened the wound and removed the ligature. At first, no change was demonstrated. Twenty hours after the release of the ligature, however, there appeared necrosis of many of the tubule cells in areas supplied by the ligated vessel. It is only after occlusion of much longer duration that the connective tissue of the kidney or stroma begins to show the effects of ischemia. This undoubtedly occurs in cases in which a branch of the renal artery has been permanently occluded, because in our forty-eight hour specimens practically none of the circulation of the infarcted half of the kidney had yet been reestablished (Fig. 10). The response of the stroma is

Fig. 22.—Outline drawing of specimen shown in Figure 21 to show the location of the anastomotic branch described.

more delayed, but the effects of connective tissue hyperplasia are more severe.

This stroma normally envelops all the structures of the kidney.[21] It arises as a network of fibrils directly from the adventitia of the larger and even the smaller vessels. The finest vascular branches are enclosed in the meshes of this fibrillar network which also winds around the tubules in spirals. Here it takes part in the formation of the membrana

21. Disse, J.: Harnorgane, Handb. d. Anat. d. Mensch. **7**:87-90 (Part 1) 1902.

propria, which is really the condensed connective tissue of the kidney on which the epithelial cells lie.[22] Both Tilp [23] and Ziegler [24] have pointed out that in damage to the kidney, when the connective tissue apparatus is not disturbed, there may be a complete return to normal morphology and function, but when the stroma also is damaged, as in renal infarcts, repair is never complete. As may be seen from Foa's description,[11] even areas of active epithelial regeneration may again be completely lost through connective tissue shrinkage.

The presence of an anastomotic accessory circulation to the kidney was shown, in 1880, by Litten.[20] He observed that if the renal artery and then the renal vein were ligated, the kidney would nevertheless slowly distend with blood. Lindemann [25] later demonstrated anastomoses between the perirenal and perforating arteries of the kidney and the suprarenal, lumbar and internal spermatic arteries. These anastomoses he found to be present, however, only in those kidneys in which a hydronephrosis had been produced by ureteral ligation. MacNider has seen in his section of infarcted cat's kidneys a collateral circulation developing as vascular streaks between the arteriolae rectae and the pseudo-arteriolae rectae of the ligated and unligated areas.

That such a collateral circulation is unable to keep an infarcted area in a functioning state is beyond question. The resulting massive necrosis indicates that the tissues are unable to remain alive.

The reason for this extensive and early necrosis can be seen from an examination of the protocols and illustrations. The tiny anastomotic branches which normally occur between the anterior and posterior portions of the kidney through the vessels of the pelvic wall and ureter are at first too small in caliber to carry a supply of blood sufficient to maintain life in the infarcted half. Even after forty-eight hours, the channels have not become wide enough to send a stream of appreciable size into the arteries of the infarcted region. The subsequent increase is more rapid. In seventy-two hours, the primary branches of the infarcted area are seen to be fairly large, and many small branches appear in the cortical zone. The damage to epithelium and stroma, however, has already occurred as the result of the long period of anemia.

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22. Rühle, G.: Ueber die Membrana propria der Harnkanälchen und ihre Beziehung zu dem interstitiellen Gewebe der Niere, Arch. f. Anat. u. Physiol., Anat. Section, 1897, pp. 153-170.

23. Tilp, A.: Ueber die Regenerationsvorgänge in den Nieren des Menschen, Jena, 1912.

24. Ziegler, E.: Lehrbuch der allgemeinen Pathologie und der Pathologischen Anatomie, Jena **2**:836, 1906.

25. Lindemann, W.: Ueber Veränderungen der Vascularisation der Niere bei Harnleiterunterbindung, Centralbl. f. allg. Path. u. path. Anat. **6**:185-186 (March 14) 1895.

While epithelial regeneration is beginning in scattered areas, connective tissue overgrowth is already in progress and will soon involve the entire area in a rapidly shrinking scar. In five weeks, the anastomotic channels to the infarcted portion of the kidney have become larger, and the arteries in the infarct closely resemble the normal side in appearance

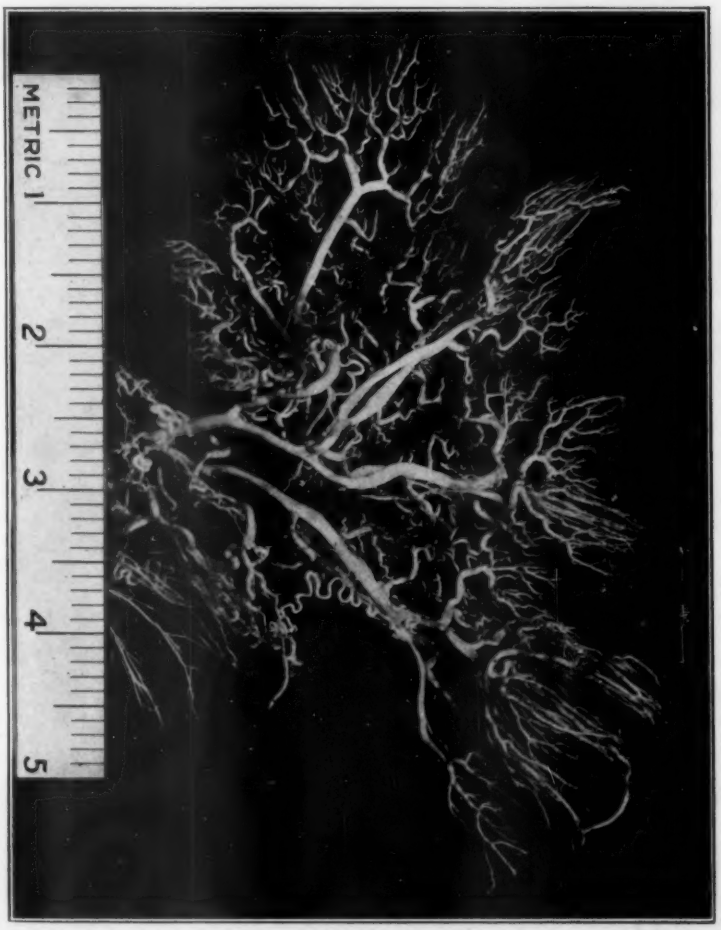

Fig. 23.—The separated infarcted portion of the injected specimen of a dog's kidney seven weeks after severance of the anterior division of the renal artery. The vessels are of fairly good size and well filled, but no glomeruli are present (comparison should be made with Figures 19 and 20). The uninfarcted posterior portion showed the usual rich supply of glomeruli like that seen in Figures 6 and 17.

and in the arrangement of their finer branches. All the finer branches show great tortuosity, however, and glomeruli are infinitely less numerous than on the normal side. This is the period of maximal

epithelial regeneration.[26] In an infarct of seven weeks' duration, marked regression of this circulation is revealed. The finer branches are shorter and are shorn of glomeruli. Such an infarcted area is the center of a depressed stellate scar overhung by the edges of the parenchyma of the opposite side.

SUMMARY

1. By means of the celluloid injection method, studies were made of the arterial circulation of human and dog kidneys.

2. As pointed out by other observers, the presence of a definite endarterial circulation in the parenchyma was readily demonstrated. Anastomoses, however, were observed between the fine arteries supplying the pelvis and ureter and between glomerulus bearing vessels which had perforated the capsule and supplied areas of perirenal fat.

3. Ligation of one of the main renal branches before injection of the kidney resulted in a punched out area which contained none of the injection mass, and which was not supplied through any anastomotic branches from the injected renal vessels.

4. Human kidneys, in which an aberrant vessel furnished part of the arterial supply, were injected with a celluloid solution of one color through the aberrant vessel and a contrasting color through the main renal artery. The main arterial supply and the polar supply were found to be independent of each other and not connected by any anastomotic branches.

5. Dog kidneys in which experimental infarcts were produced by ligating the anterior division of the renal artery were also injected. Injections were made forty-eight hours, seventy-two hours, one week, two weeks, three weeks, five weeks and seven weeks after the production of the infarct. In the forty-eight hour specimens, only a very few and scant vessels appeared in the infarcted area. In seventy-two hours, there was a marked increase in the number and size of the larger vessels of the infarct. In a week, numerous finer branches also appeared. In two weeks, glomeruli began to appear. The three and five week specimens showed an increase of glomeruli in the infarcted area, but their number was infinitely less than that of the normal portion of the kidney. At seven weeks, the glomeruli had again disappeared. Anastomoses between the posterior tree of vessels and the anterior ligated tree took place through pelvic and ureteral arteries, perforating arteries, and small intrarenal arteries which ran directly between the larger vessels of the two trees. However, these anastomotic channels were very small and not numerous.

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26. MacNider (Footnote 12, second reference).

CONCLUSIONS

The changes produced in the circulation of the kidney following ligation of one of the larger renal arterial branches are clearly portrayed by means of the celluloid injection method. An almost absolute ischemia confined to the region supplied by the blocked vessel is followed by a late ingrowth of small anastomotic channels into the bloodless area. These increase in size up to the fifth week, after which scar tissue formation, resulting from the effect on the stroma of the long period of ischemia, again makes the area relatively avascular. The end-result is a depressed stellate scar equal in area to the region supplied by the ligated branch.

The clinical significance of this is self-evident. Although, in renal surgery, it may at times be necessary and justifiable to divide one of the larger branches, it should be borne in mind that such a procedure causes a necrosis of the area of renal parenchyma supplied by that vessel. While this necrosis may not be of a magnitude which will result in clinical evidence of renal insufficiency, still, it does definitely decrease the amount of renal reserve tissue, for restoration of function in such areas is shown to be anatomically impossible.

ECTOPIC ADENOMYOMA: POSTOPERATIVE INVASIONS OF THE ABDOMINAL WALL*

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So many cases of adenomyoma occupying unusual locations beyond the uterus have been described that their presence in extra-uterine tissues is no longer considered remarkable. Such tumors have been discovered in the uterine adnexa, in the ligaments supporting the uterus and in the tissue separating the generative organs from contiguous structures. Besides the adenomyomatous tumors in these adjacent structures, others are found in tissue as far removed as the intestinal tract, the gallbladder and the kidney. It is true that there are certain distinguishing characteristics between the latter tumors and those of the uterus, especially with regard to the character of the epithelium they contain.

No such distinction can be made as regards the tumors involving the abdominal wall that are described in this report. They are of rare occurrence and consequently no considerable number have been reported. We have observed nine patients who developed such tumors of the abdominal wall following various operations on the pelvic organs.

REPORT OF CASES

CASE 1.—A woman, aged 46, had for one year noted a gradually growing lump in the site of a scar from a laparotomy performed elsewhere for ventral suspension. A hard mass, movable with the abdominal wall and connected with the fundus of the uterus, was found lying under the operative scar, midway between the pubic bone and the umbilicus.

At operation, a tumor was found extending down into the right side of the abdomen and adherent to the abdominal wall, which was diagnosed pathologically as diffuse adenomyoma. Because of the large size of the tumor, no attempt was made to remove it completely. Repeated treatments by radium have somewhat decreased the size of the tumor. This is the specimen in which the histologic tubercle was found on microscopic examination.

CASE 2.—A woman, aged 35, complained of having had tenderness in the lower abdomen, especially during menstruation, since ventral suspension and fixation of the uterus for prolapse had been performed, four years before. A hard, irregular mass was found below the laparotomy scar and adherent to the fundus of the uterus.

At operation, a mass, 5 by 2 cm., attached to the fundus of the uterus, was excised from the abdominal wall. The specimen proved to be a typical adenomyoma. The uterus was brought forward by intraperitoneal shortening of the round ligaments.

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CASE 3.—A woman, aged 38, had noted soreness in the pelvis, always worse during menstruation, following an internal Alexander operation elsewhere, four years before. A mass in the laparotomy scar likewise became tender during menstruation. On examination, the mass was found to be adherent to the fundus of the uterus.

At operation, there was a collection of dark fluid in the abdominal wall at a point where the uterine ligaments had been sutured together over the rectus muscles for suspension of the uterus. A subtotal abdominal hysterectomy was performed, and the mass in the abdominal wall excised. The histologic picture was that of an adenomyoma.

CASE 4.—Twelve years previously, a woman, aged 43, had a ventral suspension performed elsewhere because of pain in the small of the back, thought to be due to retroversion of the uterus. Eight years later, a second laparotomy was performed elsewhere and the uterus was fixed to the abdominal wall. One year later, the patient noted a tingling sensation and slight pain in the laparotomy scar, which were always worse before and during menstruation. Examination disclosed a hard irregular mass, which was not tender, under the laparotomy scar. The uterus was apparently in acute anteflexion and probably fixed to the mass.

At operation, the fundus of the uterus was densely adherent to the abdominal wall below the mass. A subtotal abdominal hysterectomy was performed, the mass and the uterus being excised in toto (Fig. 1). Histologic examination revealed adenomyoma.

CASE 5.—A woman, aged 30, had had the uterine ligaments shortened elsewhere for prolapse five years before. Three years later, a lump was noted in the abdomen beneath the laparotomy scar, which became painful following menstruation. A small mass was found in the lower end of the scar which was painful to touch, and apparently not adherent to the uterus. The patient also had had a left parotid tumor for several years.

At operation, a mass, 3.5 cm. in diameter, was excised from the lower angle of the right abdominal scar. The mass extended through the muscle and abdominal wall and was attached to a point on the left tube about 3.5 cm. from the horn of the uterus. Microscopic examination disclosed adenomyoma.

CASE 6.—A woman, aged 27, had been seen at the Mayo Clinic twelve years before, when she had had a lacerated perineum and cervicitis. The uterus was normal in size, freely movable and anteflexed, and operation was not advised. Four years before, the cervical laceration had been repaired and a tube and hemorrhoids removed elsewhere. Two years later, an operation for "adhesions" was performed elsewhere, because of a ridge in the lower right abdomen which caused a burning sensation. Her complaint on admission was a feeling of an occasionally tender mass in the right lower abdomen. Examination revealed a mass in the laparotomy scar, thought to be a desmoid. It was tender to pressure and adherent to the fundus of the uterus. Masses from the abdominal wall and the right horn of the uterus were excised. On pathologic examination, they were found to be diffuse adenomyomas.

CASE 7.—A woman, aged 36, had had curettement and ventral suspension performed elsewhere three years before. She had noted slight residual tenderness in the wound six weeks after the operation, and shortly thereafter a swelling in the lower end of the wound, which gradually became larger, was occasionally tender and intermittently discharged a serosanguineous fluid. Examination revealed an indurated area beneath the old scar, with a nodule to the right. The uterus was movable.

At operation, a mass was found in the abdominal wall, approximately 3 cm. in diameter, and a small fibromyoma was excised from the fundus of the uterus. Pathologic examination revealed adenomyoma.

Case 8.—A woman, aged 40, had had an oophorectomy and abdominal hysterectomy performed elsewhere six years before. Recently, she had noted a mass in the abdomen which, on examination, appeared to be coming from the pelvis, and felt cystic.

Fig. 1.—Adenomyoma of the abdominal wall, adherent to the fundus of the uterus. Scarring of the fundus uteri and extension of the adenomyoma into the corium (Fig. 4) may be noted.

At operation, right oophorectomy and salpingectomy were performed for an ovarian cyst, approximately 15 cm. in diameter, and a mass was excised below the previous laparotomy scar, approximately 3 cm. in diameter, which proved to be an adenomyoma.

Case 9.—A woman, aged 35, had had bilateral salpingectomy, right oophorectomy and appendectomy performed elsewhere nine years before. The wound

drained for three months and had to be resutured. Six years later, the patient had noted, in the site of the old operative scar, a small scarlike tumor apparently growing larger. It was painful and larger during menstruation. Examination revealed a tumor, 2 cm. in diameter, extending downward to the lower angle of the operative scar.

At operation, the tumor in the abdominal wall was found to extend down to the peritoneum with the omentum adherent to its inner surface. There was no demonstrable connection between the uterus and the tumor. The mass was excised and proved to be an adenomyoma.

PATHOLOGY

The gross characteristics of adenomyoma of the abdominal wall are as clear cut as those of the wall of the uterus. The largest in our series was 6 by 4 by 4 cm. in diameter. They are very firm and are similar in consistency to a uterine fibromyoma. The skin and previous operative

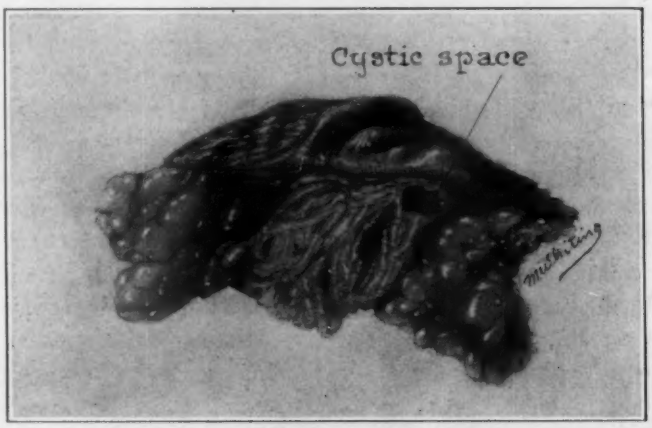

Fig. 2.—Cross-section of adenomyoma situated just below skin. Cystic space immediately below and elevating the epithelium may be noted. This cystic space could be seen below the epithelium before excision as a bluish area.

scar may be dimpled from the invasion of the tumor lying beneath the epithelium, and here and there are small dark blue or black areas immediately beneath the surface of the skin in the substance of the tumor, due to the cystlike spaces (Fig. 2).

A section of the tumor reveals grayish fibrous striated tissue surrounded by subcutaneous fat, adherent to the skin above. Whitish bands run through the tissue, between which are small punctiform openings, 1 cm. or more in diameter. These cystic spaces are glands lying between the muscle bundles, and are filled with a dark chocolate brown fluid, the remains of old hemorrhage and mucus secreted by the glands. These spaces are found in all parts of the tumor tissues. Those directly under the skin can be seen before operation as the bluish areas described.

They are usually oval, but may be elongated. The smaller cysts may be mistaken for blood vessels, especially after the tissue has been fixed and the blood in the smaller vessels becomes dark brown. With a low power glass, the lining membrane of the punctiform spaces may appear smooth and glistening white, being similar to a lining mucous membrane (Figs. 1, 2 and 3).

The stroma of the tumor is made up of fibrous connective tissue with bands of muscle which histologically resembles smooth muscle, both in appearance and in the staining reaction with van Gieson's stain. Bands of striated muscle still remain in the tumor tissue. Scattered between the stroma are gland spaces of varying size. These are lined with an epithelium which is mostly columnar, but varies from columnar to cuboidal. Some of the cells are filled with mucus and others are ciliated. The glands retain the normal appearance of uterine glands, and in many sections they are surrounded by a cellular stroma seen in the normal uterine endometrium, the so-called cellular mantel noted by German observers. In other sections, the glandular epithelium rests directly on the connective-tissue stroma. If these glands are traced through several sections, their size varies at different levels, and they follow the paths of least resistance in that whenever fibrous tissue or a muscle band is encountered, they alter their course and wind in and out in the stroma. In certain sections, the gland spaces become enlarged and form large cystic spaces, which probably are due to an obstruction caused by the pressure on their lumen from the growth and inflammatory reaction in the surrounding tissue. These cystlike spaces contain desquamated epithelial cells, large mononuclear, phagocytic cells filled with blood pigment, lymphocytes, occasional polymorphonuclear neutrophils and a carrying quantity of old blood pigment and cells débris. Occasionally, there are foreign-body giant cells containing granular blood pigment (Figs. 4 to 8). Scattered throughout the stroma are areas showing various stages of inflammation, many containing lymphocytes and foreign-body giant cells in great numbers, with nuclei in crescent arrangement. Old blood pigment is seen either lying between the bands of stroma tissue or in phagocytic cells. Other areas show more of an acute inflammatory reaction, with edema, hemolyzed blood and serum, polymorphonuclear neutrophils and a few lymphocytes. At the edges of the tumor tissue are bands of striated muscle, the fibers of which are in varying stages of degeneration, with clumping of nuclei resembling at first hand foreign-body giant cells.

In one of our cases, tuberculosis was present. There are fewer gland spaces than in typical adenomyomas of the wall of the uterus. Some of the spaces are not surrounded by the cellular stroma typical of uterine endometrium, but lie on the fibrous stroma. In this respect,

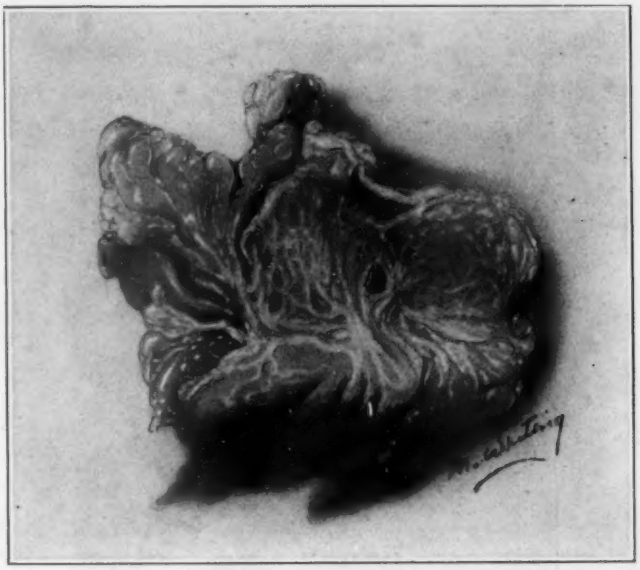

Fig. 3.—Adenomyoma showing typical fibrous striated cut surface with cystic spaces throughout the stroma. Invasion into the subcutaneous fat simulating carcinoma may be noted.

Fig. 4.—A typical histologic picture of an adenomyoma with glandular epithelium resembling uterine glands surrounded by a cellular tissue, seen also in the uterine endometrium (Fig. 1).

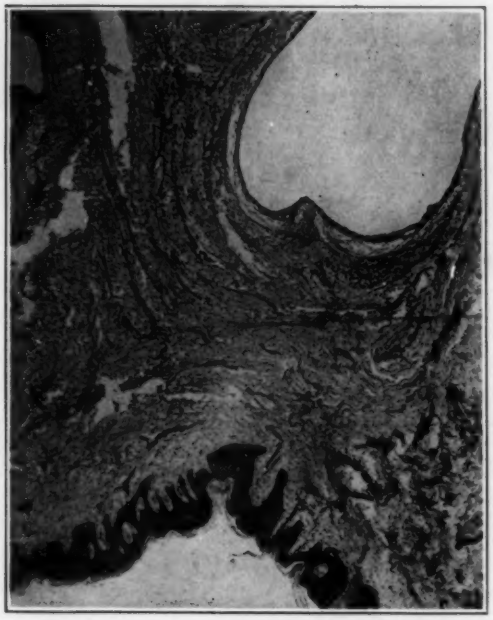

Fig. 5.—Low power photomicrograph showing the relation of a cyst space to the surface epithelium.

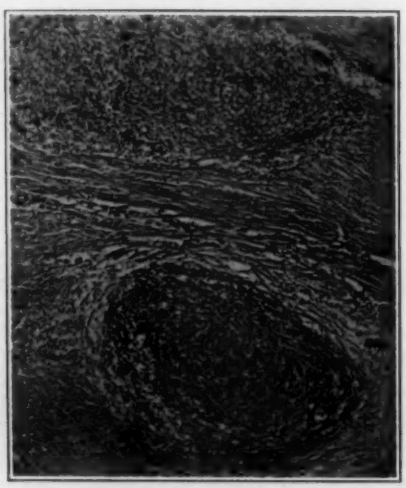

Fig. 6.—Adenomyoma of the abdominal wall associated with tuberculosis, showing typical tubercles.

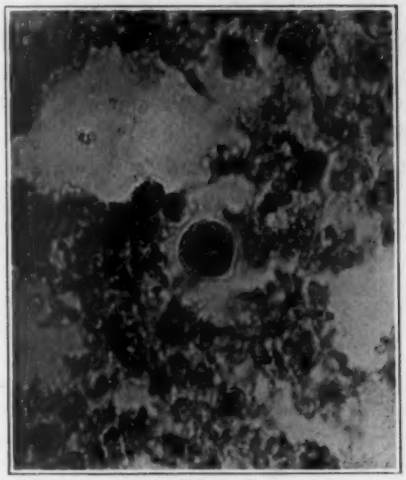

Fig. 7.—High power photomicrograph showing the exudate formed in the gland spaces. The marked degeneration of the leukocytes and erythrocytes and the large phagocytic mononuclear cell filled with old blood pigment may be noted.

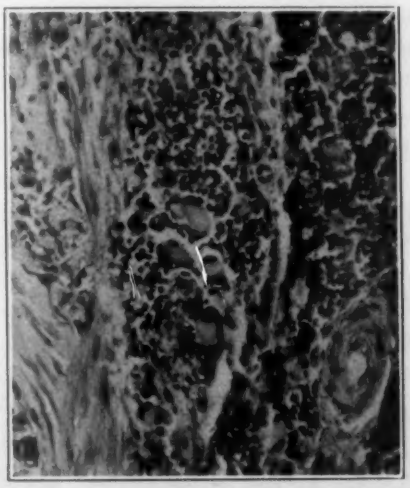

Fig. 8.—From an adenomyoma, showing a marked inflammatory reaction, great numbers of lymphocytes and foreign-body giant cells. The high power photomicrograph does not include in the field the glandular tissue that was in close proximity and identical with that shown in Figure 4.

they resemble closely the adenomyomas found in the fallopian tubes, rectovaginal septum and wall of the rectosigmoid.

In none of the cases in which adenomatous masses in the abdominal wall were attached to the fundus of the uterus were we able to find adenomyoma in the uterine wall, or to trace adenomyoma by serial sections from the uterine endometrium through uterine musculature out into the adenomyoma of the abdominal wall. It is difficult definitely to outline the adenomyoma from the fundus of the uterus, because the scar tissue appears to extend into the fundus. The scarring appeared to be the result of suturing through the fundus at the time of its fixation to the abdominal wall. Sections through this fibrous bridge showed only scarring of the musculature and no glandular tissue.

COMMENT

Adenomyomas contain smooth muscle, glandular alveoli and hyperplastic cellular connective tissue. The glandular alveoli present various manifestations. They form acini, single or in groups, and their epithelium varies from cuboidal to cylindrical, and may be ciliated. Their function simulates that of the uterine mucosa and is influenced by the variations in function to which the latter is subject.

In the series of cases observed by us, the acini have an epithelial lining identical in appearance to that of the uterine mucosa, from which it presumably had its origin, though located distantly from it. Many variations from the normal mucosa as seen in the uterus appear, not only in the morphologic characteristics of the acinal cell, but also in the character of the surrounding tissue. In fact, the cellular stroma and the epithelium itself may be present or absent, as in larger cysts. Such changes might be expected in a tumor in this remote location. In the main, however, the adenomyomas in the uterus are not unlike those in other locations, and cysts may occur in adenomyomas in any region. It is not uncommon to find malignancy in the uterus and adenomatous tumors associated, as has been shown by various authors, but in our group of ectopic adenomyomas, we found no evidence of malignancy. In one instance, the adenomyoma was associated with a tubercle typical of tuberculosis.

Recklinghausen,[1] who advanced the wolffian duct theory, believed that adenomyomas, possibly derived from the uterine mucosa, were prone to become cancerous. Lockyer [2] believed that this might be true

1. Recklinghausen, F.: Die Adenomyome und Cystadenome der Uterus und Tubenwandung, ihre Abkunft von Resten des Wolffschen Körpers. Im Anhang: Klinische Notizen zu den voluminösen Adenomyomen des Uterus von W. A. Freund, Berlin, Hirschwald, 1896.

2. Lockyer, G. L.: Fibroids and Allied Tumors, New York, The Macmillan Co., 1918.

in rare cases, when a neoplastic process was set up by chronic inflammation of a mucous membrane. In none of our cases did carcinoma develop in the adenomyoma, although the association of adenomyoma and malignancy was noted.

LOCATION

Adenomyomas of the uterus and tubes as well as those extending into the broad ligament were described before 1894 by Babes,[3] Breus [4] and Diesterweg.[5] In 1898, Recklinghausen described such a tumor situated at the base of the right labium majus, and others have been noted in the round ligament,[6] ovarian ligament, rectovaginal septum and ovary. In the ten cases reported from the Mayo Clinic in 1920:[7] one tumor was located in the umbilicus, one in the sigmoid, one in the rectovaginal septum; two tumors were in the abdominal wall, and two in the groin. Four years before, Cullen [8] described cases of adenomyoma of the umbilicus and found thirteen cases reported in the literature, four of which, however, were doubtful.

So-called adenomyomas occur in the stomach and intestines, and have been described as occurring in the kidney and gallbladder. The glandular tissue in these cases is similar to that of the tissue in which the adenomyoma exists. In this respect, the tumors vary from the adenomyomas under consideration. Finally, Ewing,[9] in 1918, described adenomyomas located beyond the uterus, in the broad and round ligaments, groin, cervix, vaginal wall, fallopian tubes and rectum, and one of us,[10] in 1921, reported twenty-three cases of adenomyoma of the fallopian tube.

ETIOLOGY

The body of the uterus, part of the vagina and the tubes are developmental products of Müller's duct. Abnormalities in these structures

3. Babes, V.: Ueber epitheliale Geschwülste in Uterusmyomen, Allg. Wien. med. Ztg. **27**:36-48, 1882.

4. Breus, C.: Ueber wahre Epithel führende Cystenbildung in Uterusmyomen, Wien, 1894.

5. Diesterweg, A.: Ein Fall von Cystofibroma uteri verum, Ztschr. f. Geburtsh. u. Gynäk. **9**:191, 234, 1883.

6. Cullen, T. S.: Adenomyoma of the Round Ligament, Bull. Johns Hopkins Hosp. **7**:112-114 (May-June) 1896.

7. Mahle, A. E., and MacCarty, W. C.: Ectopic Adenomyoma of Uterine Type (a Report of Ten Cases), J. Lab. & Clin. Med. **5**:218-228 (Jan.) 1920.

8. Cullen, T. S.: Embryology, Anatomy and Diseases of the Umbilicus, Philadelphia, W. B. Saunders Co., 1916.

9. Ewing, James: Neoplastic Diseases, Philadelphia, W. B. Saunders Co., 1919.

10. Mahle, A. E.: Adenomyoma of the Fallopian Tube, Surg., Gynec. & Obst. **33**:57-61 (July) 1921.

seem to be best explained by peculiarities in the metamorphosis of the duct into these organs, and it would seem more certain if the anatomic relationship between the normal and abnormal structures could be demonstrated. The latter condition has been fulfilled in the cases of adenomyoma of the uterus. The acinal epithelium has been traced to the uterine mucosa, and patent canals have been shown, not only in the uterine types, but also in the extra-uterine. In summary, Cullen [11] states:

> In the spaces between bundles and occasionally surrounded by circular rings of muscle, we find spaces filled with translucent and slightly punctiform tissue, areas of uterine mucosa. Sometimes its direct connection with the mucosa of the uterine cavity can be traced. Often are noted cyst-like spaces scattered throughout the diffuse myoma. These are filled with a chocolate-colored fluid and are lined with a definite membrane, often 1 to 2 mm. thick. They are immature uterine cavities and the chocolate-colored fluid is old menstrual blood that could not escape.

Cullen found fifty-five of fifty-six cases of diffuse adenomyoma of the body of the uterus, and fifty-six of seventy-three cases of all types of adenomyomas (subperitoneal, submucous or diffuse) in which the glandular elements were shown to be derived, in part at least, from the uterine mucosa. The tubal adenomas resemble the diffuse adenomyomas of the uterus in which the glands have been traced to the uterine mucosa, and are directly derived from it. Cullen believes they fulfil every requirement of normal uterine mucosa. He is not certain of the wisdom of denying that they might possibly be derived from the remains of the wolffian duct, but he thinks the evidence is overwhelmingly in favor of the hypothesis that they originated in Müller's duct.

Clinically, these growths are influenced sympathetically by the menstrual flow. They increase in size during the period and decrease with its cessation. Normal as well as adenomatous epithelioma, regardless of location, shares this function, which is possessed by no other mucous membrane in the body. In Williams' [12] case of pregnancy, the stroma of the adenomyomatous islands had been converted into typical decidua. These well known characteristics of uterine mucosa are common to adenomyomatous tumors, whether intra-uterine or extra-uterine, and no less so to the tumors that are not connected anatomically with the generative organs. In the latter tumors, the structure and the function of the epithelium are similar, and undergo the cyclic changes characteristic of uterine mucosa. In Case 4 of our series, the tumor became enlarged and more turgid during menstruation, with brownish colored cysts beneath the surface of the skin. Subjective itching and

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11. Cullen, T. S.: Adenomyoma of the Uterus, Philadelphia, W. B. Saunders Co., 1908.

12. Williams, W.: Quoted by Lockyer (Footnote 2), p. 380.

burning became sufficiently troublesome to produce excessive nervousness and insomnia. These symptoms subsided with the cessation of activity in the uterine mucosa.

THEORY OF ORIGIN IN THE ABDOMINAL WALL

It has been established that the tumors of the abdominal wall are identical in structure and cyclic function with those that are derived from the uterine mucosa, but the manner of their production is still a matter of speculation. Obviously, the hypotheses of mesonephric or wolffian-duct origin cannot be entertained, because the abdominal wall is devoid of such structures. Nor can the hypothesis of peritoneal inclusions be accepted, because, if true, adenomyomas, other than those of the uterus, would be found after operation. In our experience, such tumors have never been found in the abdominal wall of males.

Thus, the hypotheses of Weishaupt [13] and of Recklinghausen, so vigorously opposed by Kossmann,[14] whose own hypothesis was later opposed by Cullen, and by Baldy and Longcope,[15] are untenable. The conception of Cullen must be entertained because of the similarity of the tumors in structure and behavior to the uterus. The process by which the uterine epithelium finds its way to the abdominal wall, locates there and develops into a true adenomyoma, demands consideration.

Chiara,[16] in 1887, concluded that six of 760 specimens of swollen tubes which he examined postmortem were not tumors in the strictest sense of the term, but in reality products due to chronic catarrh of the genital system. In one tube, he saw signs of recent inflammation, from which he concluded that the inflammatory condition in the remaining five had subsided. Von Franque,[17] in 1900, demonstrated that tubal adenomyomas are derived from immature mucous membrane by a process of inflammation. Likewise, Meyer,[18] in 1903, found similar processes of inflammation in cases of tubal adenomyomas.

In the twenty-three cases reported by Mahle, five were associated with acute salpingitis, six with tuberculous salpingitis, one with hematosalpinx, one with hydrosalpinx, one with ectopic pregnancy and one with papillary carcinoma of the ovary. One of the patients with acute

13. Weishaupt, Elizabeth: Quoted by Lockyer, p. 320.

14. Kossmann, R.: Die Abstammung der Drüseneinschlüsse in den Adenomyomen des Uterus und der Tuben, Arch. f. Gynäk. **54**:359-383, 1897.

15. Baldy, J. M., and Longcope, W. T.: Adenomyomata of the Uterus, Am. J. Obst. **45**:788-892, 1902.

16. Chiara, H.: Quoted by Lockyer (Footnote 2), p. 307.

17. Von Franque, O.: Salpingitis nodosa isthmica und Adenomyoma tubae, Ztschr. f. Geburtsh. u. Gynäk. **42**:41-58, 1900.

18. Meyer, R.: Ueber adenomatöse Schleimhautwucherungen in der Uterus und Tubenwand und ihre pathologisch-anatomische Bedeutung, Arch. f. path. Anat. **172**:394-409, 1903.

salpingitis had been treated for gonorrheal infection six years before. It would seem, therefore, that the conditions found in inflammatory tissue are ideal for the development of adenomyomatous structures. Such tissue reaction following surgical incision takes place in the abdominal wall. Hyperemia occurs as a result of trauma to the muscle already present. It is only necessary, then, to bring the peculiarly specialized mucosal epithelium to the part to fulfil all the requirements of adenomyomatous growth. It is not necessary that the uterus or the tubes in themselves contain adenomyomatous tissue. Such a transplant is possible in bringing these structures into contact with the abdominal wall during the course of the operation. Judd's [19] explanation is logical. He believes that normal mucosal epithelium may be transplanted when the needle and suture are drawn through the uterine structures, and thence through the abdominal musculature. If this is true, the epithelium is placed advantageously for growth. This seems to be a better explanation than that in cases of adenomyoma arising in ventral fixation the gland inclusions are of peritoneal derivation, while the muscle found in the growths is the result of inflamed germ tissue, as described by Opitz. In summary, Lockyer says:

> The basis of an adenomyoma is the epithelial hypertrophy or invasion. The myomatous constituent is there because the epithelial infiltration has taken place in a structure containing muscle. Muscular tissue is not essential to the process. Epithelium will infiltrate any soft tissue during the process of repair under certain conditions. This process of repair runs riot and a branching glandular growth is produced; if muscle is the matrix in which this takes place the result is an adenomyoma.

There is nothing in this explanation that conflicts with our opinion relative to the causation of adenomyomas of the abdominal wall.

SUMMARY AND CONCLUSIONS

1. We do not believe that the accepted hypotheses with regard to the production of the adenomyomas of the uterus and tubes explain the presence of adenomyoma in the abdominal wall.

2. Cullen's hypothesis is satisfactory, provided uterine mucosa can be transplanted and will grow in the tissue of the abdominal wall.

3. Chiara's and von Franque's explanation of the growth of a transplant is plausible; and we have shown that inflammation was present in our series of cases.

4. Judd's idea that the uterine mucosa may be transplanted by needle and suture into the abdominal wall is logical.

5. Various methods of operation were performed for uterine prolapse, and in each instance it was possible for uterine mucosa to be carried to the abdominal wall.

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19. Judd, E. S.: Personal communication.

A FRACTURE CLINIC*

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NEW ORLEANS

Every clinic should possess two requirements; it must provide efficient means of diagnosis and effective methods of treatment.

In presenting this clinic for your consideration, it will be my purpose to suggest the utilization of certain neglected means of diagnosis and to urge the use of methods of treatment calculated to be of greatest service to the greatest number of the profession.

In the treatment of injuries, particularly of those in the vicinity of joints, we should avoid the diagnosis of sprain. That diagnosis should be eliminated, if by the term one understands the laceration of ligaments about joints. It is well known that the small bony prominences such as the malleoli, turbercle of the tibia and the like have less tensile strength than the ligaments, hence bony prominences are torn away from the shaft before a ligament will tear.

Many cases might be introduced here to prove the point, but I think the following will suffice:

CASE 1.—Miss McL., a trained nurse stepped from a street car, walked to her room, and the next morning reported that she had sprained her ankle. On examination, there was no swelling of the foot, but she had pain when she walked. There was localization of pain over the scaphoid. The picture revealed a fracture of the scaphoid (Fig. 1).

CASE 2.—Miss F. K., suffered from fracture of the astragalus. While running she had "turned her ankle." She walked to a theater, a distance of several blocks, limping and complaining of pain. Examination proved that there was 1 cm. difference in the circumference of the two ankles. The pain was localized over the tibio-astragaloid joint. There was limitation of all the motions of the ankle. The roentgen ray confirmed the diagnosis of fracture of the astragalus (Fig. 2).

CASE 3.—Mr. Z., while helping unload a truck, was struck on the knee. The physician treated him for a sprain and later for a "dislocated nerve." He applied to the clinic for treatment, where it was found that he had some limitation of extension of the knee, pain over the internal condyle and some hydrarthrosis of the knee. An anteroposterior view as well as a lateral view of the knee was requested, but for some reason the anteroposterior view was not taken, and a report of "negative to bone changes" was made. A subsequent lateral view was made, and this revealed a fracture of the internal condyle of the femur (Fig. 3). The loose fragment was removed, the wound healed without

* Surgical Division, Touro Infirmary.

Fig. 1 (Case 1).—Fracture of the scaphoid.

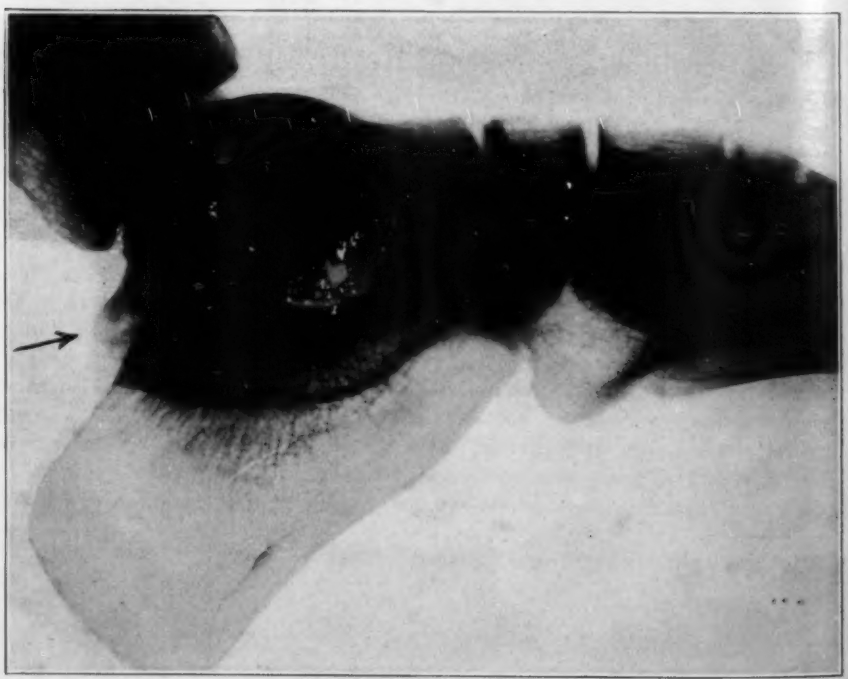

Fig. 2 (Case 2).—Fracture of the astragalus.

trouble, and the patient recovered the ability to flex and extend the knee. The last time he was seen, weight bearing was perfect, and the motions of the joint were not impaired.

A case reported once before had been that of a fracture of the greater tuberosity of the humerus, which was diagnosed as a sprain of the shoulder. The patient was not treated for a fracture until about one week after the accident.

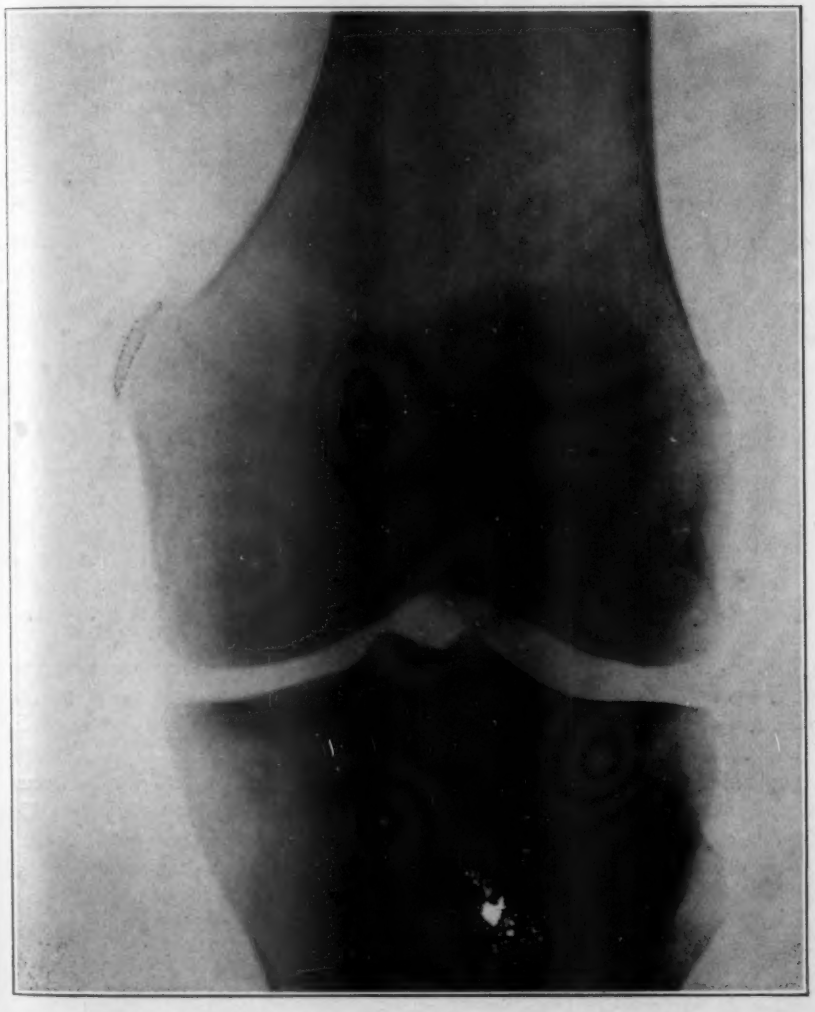

Fig. 3 (Case 3).—Fracture of the internal condyle of the femur; illustration retouched.

One could multiply these illustrations, but I think that these three cases resulting from apparently simple injuries, all of which were considered lightly by the patients for several hours, should be convincing.

A point of striking importance is that of five views of the ankle (the second case) only one revealed the fracture. From the foregoing, considered in the light of many other similar cases, one feels justified in reiterating frequently the statement that the diagnosis of sprain is not justifiable until every possible means of eliminating a fracture has been utilized.

The following cases have been selected for this presentation because a study of each and a proper interpretation of the symptoms, signs and complications present, followed by the application of the method of treatment most applicable in the particular instance, should lead one to expect good results from the standpoint of function as well as that of anatomy.

An attempt will be made, after presenting the case history, to state briefly in each instance the treatment, giving reasons for the various steps, particular stress being laid on the anatomic basis for the method of reduction and the after care, which will include consideration of the contiguous joints, necessary attention to muscles, and weight bearing.

IMPACTED FRACTURE OF THE SURGICAL NECK OF THE HUMERUS

CASE 4.—Mrs. M. M. (Fig. 4 and Fig. 5), was in an automobile accident the night of Sept. 17, 1920, and her left arm was painfully injured. She was taken to a hospital, where a diagnosis was made of a fracture about the elbow, a picture taken, and the elbow immobilized in hyperflexion. The patient came to Touro Infirmary because of persistent pain in the arm, which had not been relieved since the injury, and because of her inability to abduct or to rotate the arm. A roentgen-ray examination of the elbow was negative for fracture; the left shoulder showed an impacted fracture of the surgical neck of the humerus.

Under general anesthesia, an attempt was made to break up the impaction and correct the deformity. The arm was immobilized in plaster, in abduction and external rotation. The patient was admitted to the hospital in the service of Dr. Cohn.

September 21 a roentgenogram showed that the impaction had not been completely broken up. Under general anesthesia, the arm was manipulated by Dr. Isidore Cohn, and immobilized in abduction and external rotation with the use of the axillary triangle; traction and countertraction was made by use of the McKenna table. A roentgenogram showed perfect reduction and apposition of the fragments. The patient was perfectly comfortable and was discharged, September 25.

September 28: The patient was comfortable.

October 2: The shoulder cap was removed. There was no evidence of deformity on inspection and no swelling. The elbow was gently lifted out of the cast; the arm rotated outward gently and, with the thumb in the axilla, the head of the humerus was felt to rotate with the shaft. There was no pain. The muscles of the forearm were given rather deep massage, and light massage was used over the shoulder. The shoulder cap was replaced after padding the shoulder and arm well.

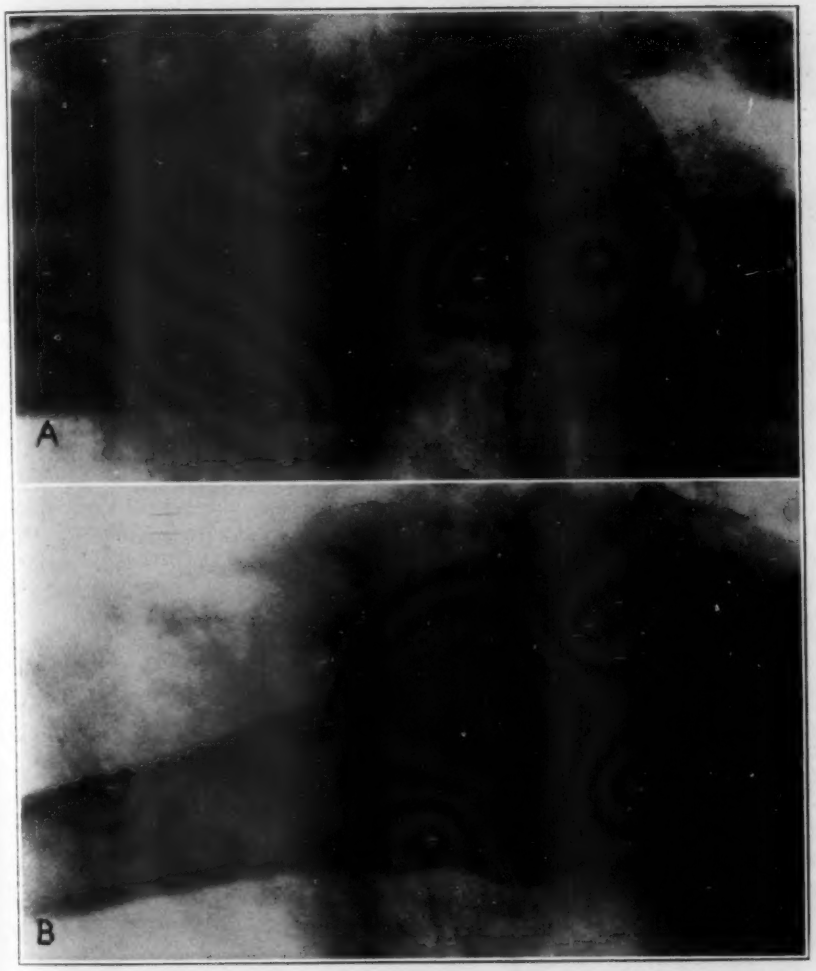

Fig. 4 (Case 4).—*A*, fracture of the surgical neck of the humerus; *B*, the same after reduction.

Fig. 5 (Case 4).—End result of fracture of the surgical neck of the humerus.

October 24: There was no deformity of the shoulder. The patient could raise the arm well beyond the horizontal position. She could place the hand on her head, on the opposite shoulder and behind her back. The measurements of the left shoulder were 36½ cm.; of the right, 37 cm. Vibratory massage was given.

October 30: The patient could abduct the arm to the angle of about 130. There was no limitation of rotation. She could put the arm behind her back without difficulty.

November 15: She was able to raise the elbow well beyond the horizontal position; external and internal rotation of the shoulder were accomplished with little difficulty, and she could put the hand well beyond the midline of the back. Measurements of the two shoulders were the same. Against resistance, she was able to lift the elbow to the level of the shoulder.

November 25: The patient was attending to her duties as a stenographer.

March 19, 1921: She said she didn't know the difference between the arms so far as disability was concerned. She could raise the arm over the head and touch both hands without difficulty. Rotation of the shoulder caused no pain, and adduction of the arm was perfect. She was able to put the arm behind her back. Resistive abduction showed a well developed condition of the muscles. With pressure against the shoulder, she was able to raise the arm to the horizontal position. The circumference of the left shoulder was 36½; of the right, 36½.

The method of treatment used in this case is typical of the method which is in common use in our service at Touro Infirmary. An anesthetic is given in all cases for the reasons that the psychic element is avoided, and muscle relaxation is obtained. Under the anesthetic anatomic restitution is attempted in every case. Manipulation and immobilization must be based on anatomic considerations. It must always be borne in mind that deformity following fracture results from several factors, one of the most important being unopposed muscle contraction. Normally, the range of motion in any particular direction is limited by the contraction of various muscles. Following fracture, this harmonious action no longer exists.

The group of muscles which are unopposed produce deformity by exaggerated contraction. In order to obtain a complete reduction by manipulation, it is necessary to bring the axis of the fragment over which you have control into the axis to the fragment over which you have no control.

This is accomplished by making your muscle pull in the direction opposite to that of the muscle contracting to produce deformity. The equilibrium thus produced must be maintained.

In the treatment of fractures of the surgical neck of the humerus, these have been the guiding principles in our work for the last twelve years.

The position utilized in the treatment of these cases is one of abduction and external rotation, with maintenance of the longitudinal traction in abduction. In the simple cases, the abduction triangle

plaster shoulder cap, and light cast including the arm and the upper third of the forearm (to maintain the external rotation and longitudinal traction), is all that is required.

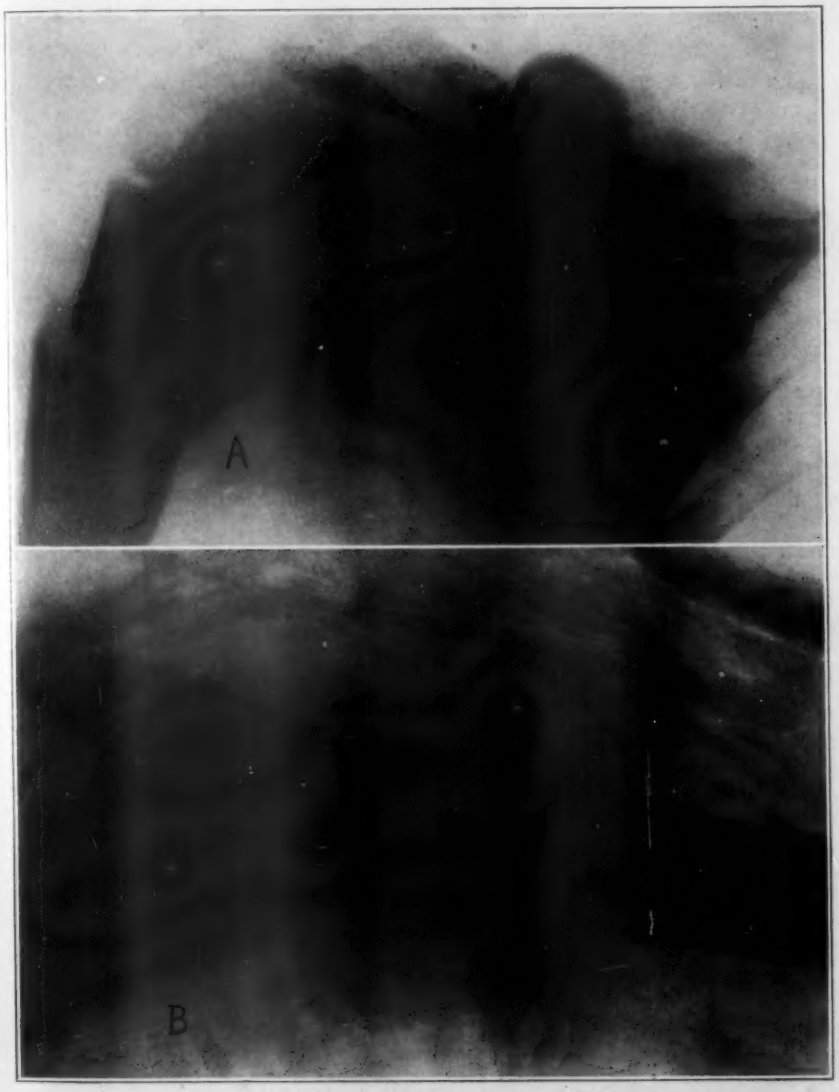

Fig. 6.—Fracture of the surgical neck of the humerus; *A*, before reduction, *B*, after reduction.

It matters not, however, whether one makes use of this particular type or whether he uses the Thomas splint, or the so-called aeroplane splint used in the recent war. The principle is the same; we must

overcome adduction and internal rotation which would be produced by the pectoralis major and latissimus dorsi. We must overcome the deformity produced by the contraction of the biceps and triceps, and we must maintain the arm in external rotation, as well as abduction, if we would bring the shaft into the axis of the upper fragment.

The patients are comfortable if the deformity has been overcome, and the bandage is not too tight. These patients are not necessarily hospital cases, except for the first twenty-four hours during which close observation should be made for interference with circulation. These patients demand close observation, if we are to expect no deformity nor limitation of motion in the contiguous joints, and if we are to expect no loss of muscle power.

These ideals may be realized by early inspection of the part. With these abduction casts, one may inspect the shoulder and the arm without altering the position of the arm. By making two parallel cuts, one on the inner aspect of the arm and one over the outer aspect, the cover of the cast can be raised without disturbing the gutter portion in the least. This we have been in the habit of doing about the tenth day. If slight movement or rotation in the gutter suggests that the early callus is not sufficiently formed, there is no further attempted motion. Manipulation is done about the end of the second week. At this time, we find that the head usually rotates with the shaft without pain. Abduction is not necessarily maintained longer than two and a half weeks, there being no longer any danger of a rotary displacement, provided there is good apposition. The joints are mobilized, from time to time; vibratory massage and contrast baths are given at each dressing, which is usually every second day for two or three weeks.

The massage is light, and the manipulation must be limited, never causing pain. The method of giving light massage by making use of the electric vibrator is so simple that one wonders that it is not more commonly used. The patients are usually encouraged to do limited active motion, with resistive exercise, about the sixth week. It is remarkable how little atrophy there is in these cases. The abscence of atrophy may be attributed to the use of resistive exercise, early motion and contrast baths, all of which help to maintain nutrition.

Nothing is more satisfying than to see these patients without deformity, and with perfect function following a routine use of a particular method.

FRACTURE OF THE RADIUS AND ULNA

CASE 5.—E. B., Sept. 10, 1920, was accidently shot through the forearm, the bullet entering about the middle third of the forearm, and passing through the shaft of the radius. The diagnosis was compound comminuted fracture of the

Fig. 7.—End result of same case (Fig. 6) ten weeks after accident.

Fig. 8.—End result of impacted fracture of the surgical neck of the humerus; this patient was not seen in our clinic during the first two weeks. The illustration is retouched, but not in the region of the fracture (same case as Fig. 9).

radius, as revealed by the roentgen ray, with marked displacement of the small fragments. There were other wounds about the patient's body, but these are not relevant to this particular injury.

Under anesthesia, the wound was debrided. The belly of the flexor sublimus digitorum had been severed by the missle. The wound edges of the muscles were debrided and sutured with mattress sutures. The wound was closed by primary suture, the deformity was reduced, and plaster splints were applied.

Roentgenograms taken subsequent to reduction showed good approximation. The dorsal splint was removed on the second day, and the wound inspected. There was no evidence of infection. The wound healed by primary intention.

Sept. 26, 1920, the head of the radius rotated with the shaft. Callus could be felt as a ring over the shaft of the radius. The roentgenogram again showed the fragments in excellent position.

October 1, the patient was able to flex and extend the fingers. There was no deformity of the forearm; the styloid of the radius occupied its normal relationship to the styloid of the ulna. Passive pronation and supination were not limited; palmar and dorsiflexion were not limited. The usual treatment, massage and contrast baths were continued.

October 16, the splints were entirely removed.

October 25, the patient could flex the wrist to a right angle. Dorsiflexion of the hand at the wrist caused some palmar flexion of all the fingers, giving evidence of some contracture. Massage, manipulation and contrast baths were given as usual. Special attention was paid to the gradual stretching of the flexor tendons.

December 23, there was marked improvement over that shown at the previous examination.

Jan. 22, 1921, the muscles of the forearm showed no evidence of deformity. The contour of the forearm was normal, and there was no angulation. The hand was capable of a good grip. Dorsiflexion of the wrist was not impaired. After resistive exercise, when the wrist was dorsiflexed, and the fingers remained extended. There was good muscle power. Resistive pronation and supination were accomplished without difficulty, showing that there was no loss of muscle power in the direction of pronation and supination.

The patient was advised to continue contrast baths and exercise.

March 3, the scar on the forearm was paler than on previous examination. Pronation was not limited, nor was dorsiflexion. The fingers remained extended when the hand was in dorsiflexion. Palpation along the forearm did not reveal any masses. The scar on the ulna side was less pale than when seen previously. Adduction caused some pain. The patient was advised to continue resistive exercise with the thumb.

May 5, dorsiflexion of the wrist was normal, as were also supination and pronation. There was evidence of good muscle tone when resistive active motion was given.

The continuation of exercise for the thumb was advised, in order that the flexor tendon might be stretched.

The patient presented many interesting problems. She had a perforating gunshot wound of the right forearm. Examination revealed the fact that some of the flexor muscles had been severed by the bullet. There were multiple comminuted fractures of the shaft of the radius. The indications seemed clearly to be, that we should

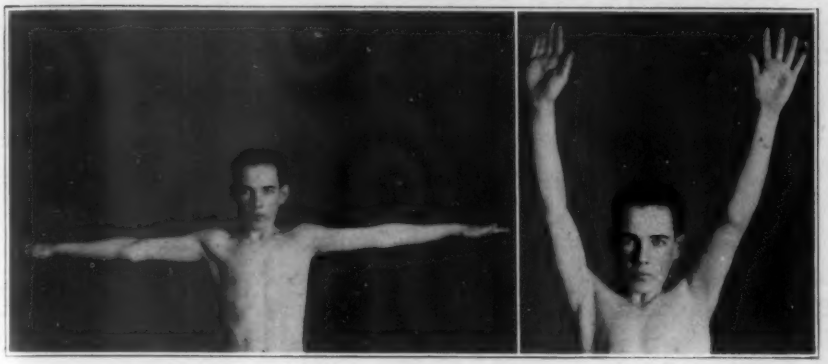

Fig. 9.—End result of impacted fracture of the surgical neck of the humerus. History: During the first two weeks following the accident no attempt was made by the surgeon in charge to reduce the deformity, as he said it was useless to expect a good result. It was at this stage he came under observation. Treatment: The deformity was reduced, abduction axillary triangle and shoulder cap applied, and external rotation of the arm maintained by a light plaster cast extending down to the middle third of the forearm.

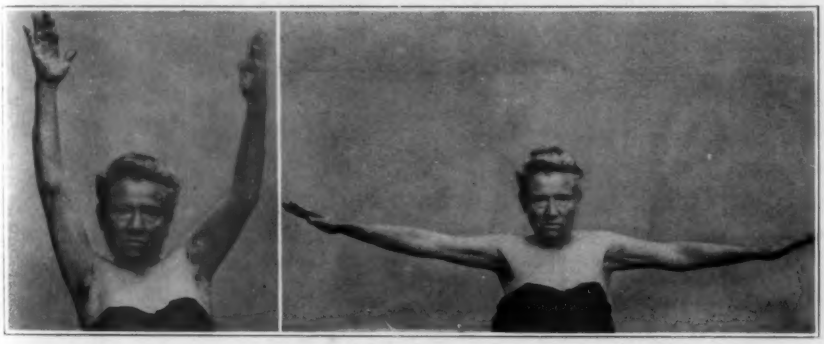

Fig. 10.—End result of impacted fracture of the surgical neck of the humerus. Treatment: Impaction was broken up, and the usual axillary triangle and external rotation of the arm employed.

Fig. 11.—End results of impacted fracture of the surgical neck of the humerus. Treatment: Impaction was broken up, and the usual axillary triangle and external rotation of the shoulder employed; longitudinal traction was maintained with the usual plaster dressing.

debride the wound edges, freshen up and approximate the severed ends of the muscles to restore the normal continuity of these muscles. This was done and the wound sutured. The fracture was then treated as a simple fracture.

The small comminuted fragments of the shaft were not disturbed, because as long as the fragments are in contact with the shaft or with the periosteum, they may act as ossifying centers, thereby favoring a more rapid repair of the bone. Vascular supply and bone contact are the only necessary factors for active participation by the fragments in repair. In the event that infection had developed within the wound, these fragments might have acted as foreign bodies and would have had to be removed.

The picture taken after reduction, showed these small fragments to have been almost entirely in the normal continuity of the shaft; further subsequent events proved the correctness of the treatment, because the wound healed by primary intention. A firm union occurred without any of the fragments being exfoliated.

Pronation and supination became normal; the patient had no bowing of the forearm, and all movements of the wrist were normal. The only disturbing feature which developed was an inability to maintain the fingers extended when the wrist was dorsiflexed. This I believed to be due to scar tissue formation at the site of the union of the muscles sutured. By the use of resistive exercise, this disability was overcome.

At present, the muscle power in the forearm and the hand is normal and the same as that on the opposite side.

This case illustrates the value of primary suture and of treating comminuted fractures, when possible, as simple fractures.

FRACTURES OF THE ULNA

Fractures of the ulna are of interest for the following reasons:

1. An improper interpretation of a normally developing olecranon may be mistaken for a fracture. This has often happened in our experience.

2. A fracture may be mistaken for a normal olecranon if one is not careful.

3. Reduction of the deformity in fractures of the upper third of the ulna is essential; if this is not accomplished the ulna acts as a pendulum, swinging as a rule away from the midline, stretching the internal lateral ligament, thus diminishing the carrying angle. More than this happens; flexion of the upper fragment, which results from contraction of the brachialis anticus, added to the change in the axis of the upper portion of the shaft alters the relationship of the sigmoid

cavity of the olecranon and the articular portion of the humerus. The result of this, if uncorrected, is a limitation of the flexion of the elbow.

Fractures of the upper third of the ulna had best be treated by hyperflexion of the elbow.

FRACTURES OF THE BASE OF THE RADIUS

No fracture clinic would be complete without mention of fractures of the base of the radius. In this type of fracture, impaction or overriding deformity results in most cases. If the existing deformity is not corrected and the normal relationship of the styloids restored, there will be a permanent limitation of flexion at the wrist. Normal function at the wrist is dependent on the maintenance of the normal axis of the radius. Change in this axis alters the articular surface relationship with the carpal scaphoid and semilunar. In turn they act as a wedge, thus limiting flexion.

Case 6.—While working at some electrical work, Monday, May 5, a monkey wrench slipped in some way, and Mr. B. fell forward, the weight of his body being transmitted on the forearm of the left arm. He was brought to Touro Infirmary, where I saw him three days after the accident. At that time, there was a marked deviation of the hand on the radial side, and angulation of the forearm. The radial styloid was on a level with that of the ulna.

The patient was given general anesthetic by Dr. Smith, reduction was carried out in the usual way, and a plaster molded splint applied.

The case was diagnosed as comminuted impacted fracture of the base of the radius, and linear fracture of the shaft of the radius. There was a small transverse fracture of the styloid of the ulna, with displacement. A roentgenogram taken after reduction showed about two-thirds of the styloid of the radius below the transverse Skinner's line.

The patient was seen in the office, May 7. There was a little swelling of the fingers. He had had no discomfort, and was able to move the fingers at will.

Roentgen-ray examination revealed a comminuted, impacted fracture of the lower end of the radius and a fracture of the styloid process of the ulna. There was a displacement backward of the carpal bones, producing a marked deformity of the wrist.

An observation made by Dr. Samuel after reduction showed that the fracture was in a good position.

July 23, 1919, pronation and supination were unimpaired, as well as abduction and adduction at the wrist. Dorsiflexion was normal. There was a slight limitation of palmar flexion, but no pain or deformity at the wrist.

October 11, five months after the injury, the patient said he was going about with his regular work and really worked as if he had never had a broken arm. He was using an electric drill weighing about 40 pounds (18.2 kg.), which he could raise and hold in position that other men might work with it.

The measurements of the wrists were: right, 17 cm.; left, 17½ cm. Abduction and adduction of the hand were unimpaired and there was no deviation. Pronation and supination were normal, and palmar flexion was only slightly limited. Dorsiflexion was unimpaired.

The foregoing case is interesting, because the method of reduction and treatment of an impacted fracture of the base of the radius is illustrated here.

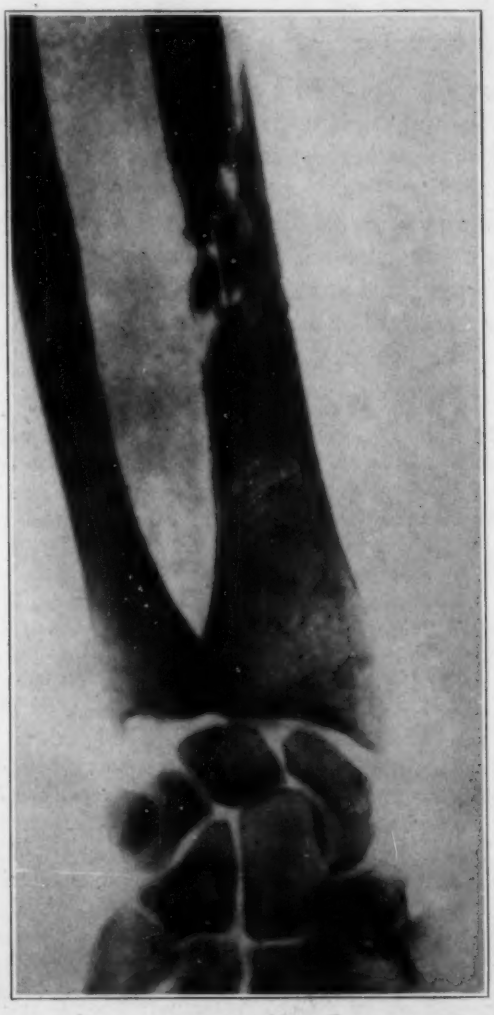

Fig. 12.—Compound comminuted fracture of the radius. Treatment: Débridement, suture of the tendons, closure of the wound, and reduction of deformity. The fracture was treated as simple fracture. End result: perfect function and no deformity.

Anesthesia is resorted to in all cases, as well as reduction by the breaking up of the impaction and applying plaster molded splints to the palmar and dorsal aspect of the forearm.

By resorting to Skinner's line, evidence of reduction is obtained. A plane is passed through the middle of the long axis of the shaft

of the radius. Intersection of this plane at right angles by a plane passed through the tip of the styloid of the ulna, if reduction has been complete, finds more than one-half the base of the radius below it.

Fig. 13.—The illustration shows the pendulum action present when deformity has not been reduced in fracture of the upper and middle third of the shaft of the ulna.

Fig. 14.—End result in fracture of the ulna where the deformity has been reduced.

An incomplete reduction will result in permanent limitation of palmar and dorsiflexion due to the mechanical limiting action of the base of the radius through its overhanging tip on the carpal bones of the first row.

Incomplete reduction is manifest clinically in the deviation of the hand to the radial side and the persistent prominent ulnar styloid. In no type of fracture is complete reduction of deformity more imperative. In no type of fracture is anesthesia more necessary.

Plaster molded splints should be substituted for all types of board splints.

Board splints, either straight or those manufactured and sold in sets, cannot be made to fit the individual. To make them sufficiently snug to immobilize, they must be tight enough to interfere in some way with circulation. Interference with circulation means atrophy, and, to say the least, atrophy means loss of muscle power; to restore this, there may be a long while during which the individual's earning capacity is diminished, thus envolving economic loss. Good results can be obtained by early complete reduction, molded splints, and early and frequent removal of splints or contrast baths, massage and passive motion.

FRACTURES OF THE FEMUR

In approaching cases of fracture of the femur, one is confronted with traditional ideas which are deeply rooted in the minds of many.

One still hears the opinion expressed that no attempt should be made to secure an anatomic restoration in old persons. It may not be put in those words, but it is stated that an impaction should not be disturbed.

At the outset, one should remember that normal function is dependent on anatomic restitution.

Some are afraid of "hypostatic pneumonia," if the aged are placed in a plaster cast or otherwise kept in bed. It is well to remember: (1) that most of the deaths are due to fat emboli; (2) that, with a properly applied cast, the patient is not necessarily confined to bed, nor must he lie in one position.

At present, there are only three recognized treatments for fractures of the neck of the femur, and all are based on the fundamental principles as outlined by Royal Whitman, with which all are familiar.

The methods are:

1. Conservative treatment using a plaster cast for purposes of immobilization after reduction of the deformity.
2. Suspension and traction.
3. Operative treatment.

My own preference is for the cast in simple, uncomplicated cases.

There are only a few practical points that I should like to direct your attention to: (1) One should always take a picture of the uninjured hip for the purpose of determining the amount of impaction;

(2) weight bearing should not be permitted for several months (I prefer to use the brace for a period ten to twelve months following the accident); (3) with the proper change of casts, mobilization of the knee and ankle does not become the serious difficulty that some would have us believe.

FRACTURE OF THE SHAFT OF THE FEMUR

Treatment of these cases may be discussed under three heads.

1. Conservative nonoperative methods.
2. Suspension and skeletal traction.
3. Operative methods.

Conservative nonoperative treatment will give equally as good results as any other method when proper attention is given to detail. One is reminded in this connection of the difference in results obtained by the home physician in the case of a business man who had a cardiac condition, and the result obtained in a health resort. At home, the patient was unwilling to observe details; he did not have time. When the patient was under the care of the physician in a health resort, his whole time was given to obeying orders.

If the same scrupulous care is given in nonoperative cases as is given in operative cases, if due attention is paid to the after care of muscles and joints, the results will satisfy the most fastidious.

In order to obtain good results, a proper traction table must be at hand. The portable roentgen ray is almost an essential in this work, and after reduction a snug fitting cast should be applied.

The cast must not be left on longer than three or four weeks. The knee is then moved and massage started. At the end of six to eight weeks, the casts are removed and a nonweight-bearing brace, which has a leather molded jacket for the thigh, is worn by the patient. Constant attention is given to the knee and ankle. Three months after the accident, I have not noted any limitation of motion in the knee and ankle. The brace must be worn seven or eight months.

When reduction can not be accomplished by traction on a table, continuous skeletal traction is indicated. The tongs of the Edmonton type are invaluable for this purpose.

This permits immediate mobilization of the knee and ankle, as well as earlier massage.

For the utilization of this method, a trained corp of assistants is needed, and the patient must be hospitalized for a long period.

This is not always feasible. What we want are good results which can be obtained by the man who can not hospitalize his patients.

INTRA-ARTICULAR FRACTURE OF KNEE; LINEAR FRACTURE OF THE TIBIA

CASE 7.—While Mr. T. was cranking an automobile, May 5, one of his fellow workmen, who was in the car, did not notice that the car was in gear, and the motor suddenly started, pinning the patient between the car and the side of the garage. He was brought to Touro Infirmary immediately.

The examination revealed swelling of the knee, deviation of the leg from the midline, fluctuation of the knee joint, and tenderness over the upper portion of the tibia. No further effort was made to examine the patient until he was anesthetized. The knee was aspirated and a large quantity of blood obtained. This fluid was sent to the laboratory for examination. No growth was obtained from the culture. An attempt was made to reduce the deformity, and a plaster cast was applied from the foot to the junction of the upper and middle of the thigh.

Fig. 15 (Case 7).—Intra-articular fracture of the tibia; *A*, before reduction; *B*, after reduction. The end results were perfect function and no deformity.

A roentgenogram taken prior to reduction showed a linear fracture of the shaft of the tibia through the head of the tibia into the joint. A wide separation existed between the fragments.

The roentgenogram, taken after reduction, showed an excellent position, with a slight break in the continuity of the head of the tibia. It was decided to attempt to approximate the two fragments more closely; in order to do this, two lateral bars were made, each containing in its middle a leather pad attached to a screw through a hole in the bar. This bar was incorporated in the cast, and in this way pressure from both sides toward the middle was obtained. The result of the use of this screw pad is shown by the roentgen ray.

May 21, the cast was removed; massage and passive motion were accomplished through an arc of 30 degrees. A light cast was applied, including the pad on either side of fracture.

May 25, the cast was again removed, the patient was given a bath, and the knee was mobilized about 45 degrees. A plaster splint was applied. From this time on, the plaster splint was removed each day. The knee was baked, mas-

saged and mobilized. The patient was able to flex the knee to 130 degrees. No swelling of the knee was noted. A nonweight-bearing brace of the Thomas ring caliper type was applied.

By June 20, he was walking without crutches, was able to flex the knee to 90 degrees and to extend it completely. There was no deformity or swelling of the knee.

June 28, the patient could flex the knee a little more. Massage, baking and active motion were continued. By July 1, he was able to flex the knee 45 degrees and to extend it completely. There was no swelling at this time, and the patient continued to use the brace.

July 28, the patient could flex the knee beyond 45 degrees and he is able to kneel down and touch the gluteal region with his heel. He has resumed his former occupation.

COMMENT

Weight bearing and a movable knee in these cases are the most important considerations. The weight of the body is transmitted direct from the condyle of the femur to the intact upper end of the tibia. In cases such as the one under consideration, lack of proper approximation of the fractured tibia would result in the condyle of the femur acting as a wedge, thus favoring a persistence of the separation, a stretching of the internal lateral ligament and a consequent genu valgum. Therefore every effort must be made to approximate the fragment to the shaft. One may elect to be conservative and use nonoperative measures, or he may operate.

My preference was the conservative nonoperative measures.

1. Aspiration of the knee followed by the application of a plaster cast should be employed. Whether one operates or applies a cast the knee should be aspirated. There was in this case, as in most intra-articular fractures of the knee, an accumulation of bloody fluids. If this fluid is not removed, the capsule becomes distended; this means loss of tonicity of the capsule, which favors the development of a weak unstable knee joint. Moreover, if this fluid is allowed to remain, organization of the clot may produce changes which will permanently damage the articular surfaces.

A word of caution may be of service; this fluid should be cultured for one's own information and protection. As noted in the history and shown by the pictures, we were able to approximate the fragments with the cast and modified Pierson pads.

2. The after care is of the utmost importance. Early mobilization, massage and baking are essential.

3. The nonweight-bearing caliper of the Thomas ring type should be worn for a period of not less than four to five months.

Those who argue for operative interference cannot produce better results than a stable, perfect functioning, weight-bearing knee, such as this patient has.

FRACTURE OF THE LOWER FIFTH OF THE FIBULA AND THE INTERNAL MALLEOLUS

CASE 8.—Miss E. H., June 22, 1923, was admitted from Charity Hospital, with a large posterior gutter splint. On inspection, the ankle was found to be greatly swollen, the swelling being particularly localized over both malleoli. There was an apparent deviation of the foot from the midline (pronation) and apparently, a slight angulation of the leg.

Palpation revealed a localized pain over the internal malleolus. There was some limitation of plantar and dorsiflexion and pain over the astragalus.

The roentgen-ray picture revealed a fracture of the fibula about 1½ inches (3.7 cm.) above the malleolus and a fracture of the internal malleolus. Lateral displacement of the fragment of the fibula was present.

Fig. 16 (Case 8).—*A*, fracture of both malleoli, prior to reduction; *B*, same, lateral view.

The patient was given an anesthetic by Dr. Allgeyer, and the ankle was manipulated and placed in supination, with the foot at right angles to the leg. A light plaster boot was applied. The roentgen-ray picture showed a complete reduction. There being no swelling, the patient was allowed to go home.

July 3, the plaster cast was removed, and a great deal of swelling was noticed over the malleoli. A marked amount of ecchymosis existed above the external malleolus. Some ecchymosis was located below and behind the external malleolus. The foot was then given a bath, heat applied with the thermolite, after which the position of extreme supination was changed to one of right angle flexion of the ankle. A light cast was applied in the new position.

July 16, the cast was removed. There was much less swelling and ecchymosis than on the previous occasion. There was no evidence of deformity. Dorsi and

Fig. 17 (Case 8).—*A*, after reduction; *B*, same, lateral view.

Fig. 18 (Case 8).—Results nine weeks after the accident. There was perfect functioning of the ankle and no deformity.

plantar flexion were slightly limited, but not painful. Pronation and supination of foot were not painful. There was no pain over the malleolus. The usual treatment was given; passive and active motion, slight resistive exercise, bath and reapplication of the cast.

July 28, there was no pain. The swelling was reduced, and there was no limitation of motion. The patient walked into the office without either crtuch or stick. The cast was removed. Resistive exercise was given and the thermolite was used. A brace was tried, but it needed a little alteration, so was returned to the shop. The advice given to the patient was as follows: She was to wear the brace constantly, take hot and cold baths, and exercise the ankle by using the sewing-machine pedal.

July 30, there was less swelling than on previous examinations, no limitation of motion in the ankle, no abnormal mobility and no evidence of deformity. The patient walked without the use of the cane, with her brace. The usual treatment was given; thermolite resistive exercise, active and passive motion. The patient was to continue her home treatment and return once a week for observation.

August 3, there was no pain; the patient was walking on a brace, and there was less swelling. Improvement was noticeable in muscle tone, as evidenced by resistive exercise. At the present time, the ankle is normal in every way.

COMMENT

There is no fracture in which complete reduction of deformity is more important than in fractures about the ankle. Weight-bearing is dependent on the maintenance of the normal mortise of the ankle. The astragalus must remain in a fixed position by the two malleoli; any thing permitting increase in the intermalleolar distance permits an alteration in the axis of the astragalus; such an axial rotation results in interference with weight-bearing and produces pain.

E. H. Skinner has shown that a plane which bisects the tibia should bisect the astragalus.

Once reduction has been accomplished, a cast should be applied, with the foot in supination.

After two weeks have elapsed, the foot may easily be placed at right angles. There is no longer much danger of a recurrence of the deformity.

Passive motion, heat, massage and contrast baths are essential as they prevent atrophy, contractures and peri-articular types of ankylosis. A nonweight-bearing brace permits the patient to walk unsupported by crutches after six weeks. Such a brace should be worn at least four months.

A STUDY OF AN UNUSUAL CASE OF MYOSITIS OSSIFICANS*

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PHILADELPHIA

The following diagnostic study of a case of myositis ossificans of the lower leg has proved to be one in which the application of a definite classification has been difficult. Clinical and roentgenologic observations of the case covering a period of four years were made, but the opinions of those associated with these studies diverged rather widely, and the case was placed in a definite category only after final gross and microscopic pathologic studies could be added to the foregoing. However, although the case has been classified as one of ossifying myositis, many features that seem to represent dual or overlapping tendencies still persist, and one is confronted by the problem of explaining the relationship between two apparently distinct processes occurring at different intervals in the course of our study of the patient. Unquestionably, the borderline or overlapping diagnosis is sometimes quite unsatisfactory and frequently annoying to the clinician, who is chiefly interested in therapeutic procedures and prognosis. On the other hand, however, careful studies of these cases are quite justified by the fact that they may often shed new light on the etiologic factors concerned in an obscure process; even more information may perhaps be derived from the study of these so-called borderline cases than from those of a more or less similar type which, exhibiting well-known textbook characteristics, easily fall into a definite classification.

REPORT OF CASE

History.—B. L., a young woman, aged 34, a social service worker, was first seen in October, 1919, when she consulted Dr. R. G. LeConte about an increase in the size of the upper portion of the calf of the left leg, accompanied by very slight pain in this region. This enlargement was apparently of three or four months' duration with no definite history of recent, preceding trauma.

Physical Examination.—The patient was a well developed young woman, in excellent health, and her general physical examination was essentially negative. There was a rather symmetrical enlargement of the upper portion of the lower leg just below the knee joint, within which a large firm mass could be palpated. The mass, which seemed to arise from the lateral aspect of the head of the fibula, apparently had the consistency of bone, and it was not tender.

Roentgen-Ray Examination.—This showed an elongated, fairly well circumscribed, oval mass (Fig. 1) occupying a position in the upper third of the leg, adjacent to, but not eroding the head of the fibula or tibia. A provisional diagnosis of enchondroma was suggested.

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*From the Ayer Clinical Laboratory, Pennsylvania Hospital, Philadelphia.

First Operation.—October 17, Dr. LeConte performed a resection of the tumor mass, including the upper third of the fibula. An incision running parallel to and over the upper half of the fibula was made. A firm, projecting lip arising from the tumor mass was found to overlap the muscles on the lateral aspect of the leg to which they were firmly adherent. The mass, as exposed, seemed to be fairly well circumscribed; its major portion was fixed to the head

Fig. 1.—Tumor mass below the knee joint, adjacent to, but not eroding the head of the fibula or tibia.

of the fibula and, to a lesser extent, to the head of the tibia. An interesting feature was the location of the common peroneal nerve, which was found with some difficulty, running in a groove in the mass along its inferior surface. It had been displaced downwards from its normal location for a distance of about 3 cm. Removal of the mass was accomplished, with resection of the upper third of the fibula and without apparent injury to the peroneal nerve.

Pathologic Examination.—The specimen included the upper portion of the fibula, its head and articular surfaces, measuring in all 12 cm. in length. Along the anterolateral surface and firmly connected with the shaft for a distance of 5 cm., was a fusiform, firm, osseous mass to which were attached fibrous and muscle tags. It separated from the shaft of the fibula with ease, leaving the long bone devoid of periosteum at this point, but otherwise apparently uninvolved except for slight thinning of the shaft along the point of cleavage with the tumor mass. The mass itself was hard, yet slightly compressible.

Cross sections of it showed a thick outer rim of compact bony tissue, averaging 1 cm. in thickness, except at the point of cleavage with the shaft, where it

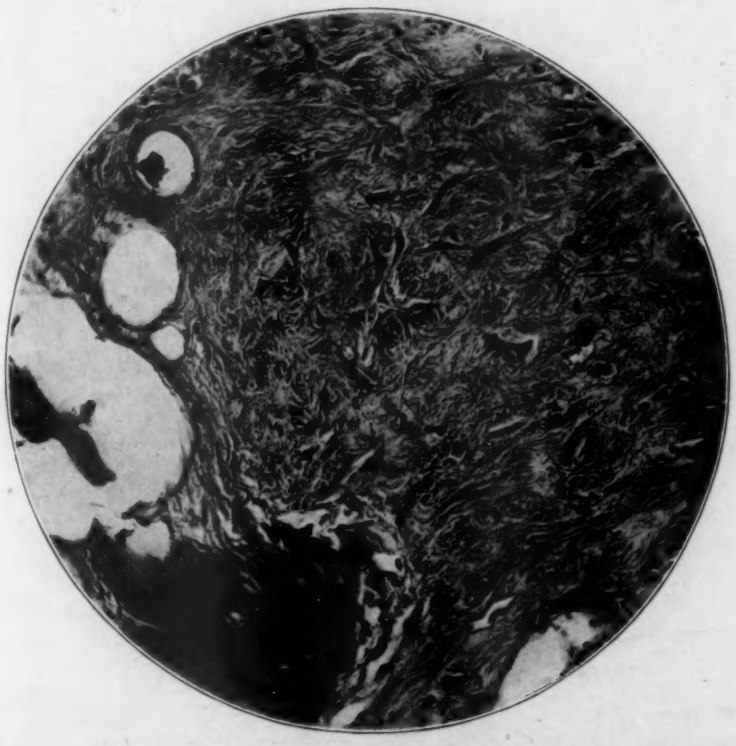

Fig. 2.—Photomicrograph (magnification 184 diameters) showing the type of tissue noted in the central portion of the primary growth, and an island of osseous tissue from the peripheral bone shell.

was much thinner. Within the outer shell, the tissue was much softer in character, cutting with the consistency of cartilage.

Microscopic Studies.—The outer bony shell, noted grossly, proved to be composed of irregular bone and fibrous tissue with its free edge abutting directly on the adjacent muscle fibers. In many places this contiguous muscle tissue appeared quite healthy, but in others it had undergone mild fibrosis and apparently subsequent ossification. Scattered about in the bony reticulum were many isolated islands of surviving muscle fibrillae. A very different picture was seen in the softer central portion (Fig. 2), or core of the tumor, where the back-

ground was quite homogeneous and composed of irregularly shaped fusiform and stellate fibroblastic cells ramifying throughout a stroma which consisted of irregularly coursing bundles of fibrillae. Here and there were isolated giant cells, evidently osteoblastic or chondroblastic in character, and more frequently small discrete islands of definite bone formation occurred, often surrounded by these same large mononuclear and multinuclear giant cells.

Fig. 3.—Recurrence in the operative area.

Diagnosis.—Sclerosing osteogenic sarcoma or ossifying fibroma of questionable malignancy was the diagnosis. On restudying these sections in the light of the subsequent findings one cannot see any particular reason for changing this diagnosis. The activity of fibroblastic cells in the central portion strongly indicated that the lesion was a tumor and should logically be interpreted as such. However, careful examination of the outer bony shell revealed an ossifying process which compares very favorably with that seen in the subsequent pathologic study.

Course.—The patient made a satisfactory operative recovery with the wound healing promptly. Unfortunately, there was no roentgenogram made immediately after operation, and there is no way of proving that all of the growth was removed. She was followed closely, but another roentgen-ray examination was not procured until a year and a half later, March, 1921. Then, the roentgenogram showed in the operative area a definite recurrence, which seemed to

Figure 4

Figure 5

Fig. 4.—Size of recurrence three years after the primary operation.

Fig. 5.—Recurrence immediately prior to the second operation. The manner in which the new growth of bone conforms to the external shape of the leg should be noted.

have some of the characteristics of an osteogenic sarcoma (Fig. 3). Numerous roentgen-ray studies were made during the next two years. By October, 1922, the recurring growth had reached considerable proportions (Fig. 4), extending

down from the knee joint to the middle of the shaft of the tibia. During this time, the external appearance of the leg had not shown any marked increase in size, although a very definite firm mass was again palpable in the upper portion of the lower leg. Slight atrophy of the muscles about the ankle was present. Roentgen-ray examinations of the chest for possible metastases were negative.

During the summer of 1922, she began to have noticeable symptoms consisting of slight pain below the knee and considerable weakness of the extensor group of muscles of the foot together with inability to rise on her left toes while walking. A roentgen-ray examination was made in December, 1922 (Fig. 5). Radical surgical procedures were deferred, owing to the prevailing opinion that the growth was actually benign; but, because of its rapid increase in size, its painful character, and the distinct influence which it was exerting upon the function of the leg, amputation was finally decided on.

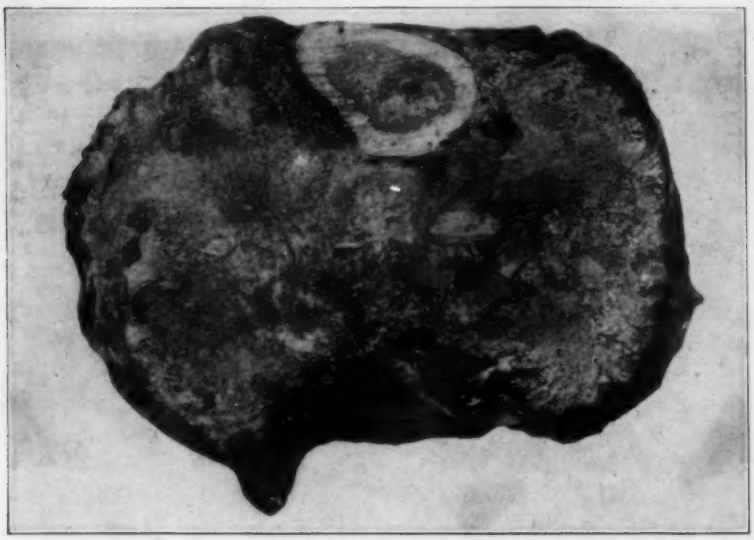

Fig. 6.—Cross section of the lower leg (junction of upper end and middle third), showing manner in which the growth of bone has conformed to the external shape of the leg and the perforating holes through which the posterior tibial vessels penetrated.

Second Operation.—Feb. 27, 1923, Dr. LeConte amputated the lower leg at the junction of the lower and middle thirds of the femur.

Pathologic Report.—The specimen included the left leg, which had been amputated 7 cm. above the knee joint. In the upper half of the lower leg there was a fusiform but rather symmetrical enlargement, which still conformed to the normal contour of the leg, starting directly below the knee joint and extending almost to the junction of the lower and middle thirds (22 cm. below the head of the tibia). Within this enlargement was an easily palpable, central mass having the consistency of bone. In several places, small bony prominences could be felt just beneath the skin where extension into the subcutaneous tissue had taken place, with adherence to the overlying skin; in no instance, however, had ulceration of the skin occurred.

On dissection of the lower leg, a large area of irregular bone formation was found (measuring 9 cm. in diameter in the upper third), occupying the entire central portion of the upper half of the lower leg and conforming to the shape of the leg. A most striking feature was the presence of this huge mass of bone within the leg and comparatively slight enlargement of the leg externally. The shaft and head of the tibia were encapsulated by the growth on its posterior

Fig. 7.—Dried specimen of bone, showing the encapsulation of the shaft of the tibia by abnormal bone.

and lateral surfaces, but it was not possible to demonstrate actual erosion of the outer table. The proximal end of the fibula (upper third resected at previous operation) was lost in the bony growth. Muscular attachments about the head of the tibia were either replaced by thin bands of fibrous tissue, or seemed to arise directly from the bony growth. Faint outlines of the muscle units in the calf of the lower leg could be traced throughout the structure of the invading

bony tissue, which was well shown on cross section (Fig. 6). An extremely interesting feature was found in the survival of some of the arteries through this mass of bone. These were present in the form of perforations traversing the central portion. The posterior tibial artery, for instance, had buried itself in the superior portion of the growth and emerged at the inferior end intact. Fluid could be injected though this artery with ease. In general, this bony mass was uniformly hard, and apparently it was entirely composed of the same type of tissue throughout. Blocks for microscopic sections were taken from several parts of the peripheral portion of the growth, and two horizontal cross sections were made. The remainder of the leg was disarticulated at the ankle

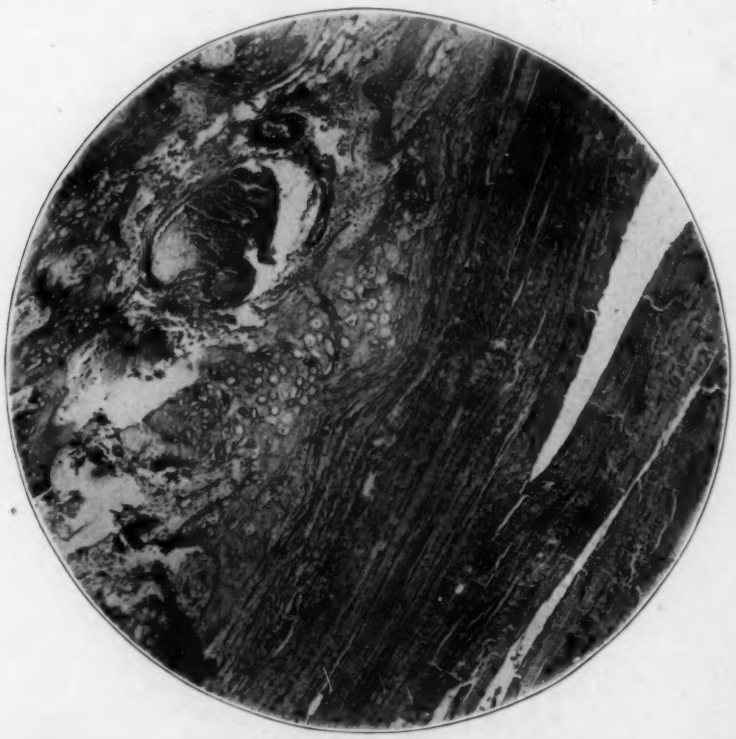

Fig. 8.—Photomicrograph (magnification 65 diameters), showing the edge of cartilaginous and bony tissue encroaching upon surviving muscle. Atrophy and evidences of metaplasia are seen in the contiguous muscle fibrillae.

joint, and a dry specimen of the bone was procured by boiling it in alkali solution (Fig. 7). The dry specimen showed clearly the ossification of individual muscle units as well as the character of the bony framework, which appeared to be laid down in a mass of fine spicules.

Microscopic Studies.—Specimens from several aspects of the main tumor growth, the surrounding tissues and the adjacent skin were studied.

These showed that practically the entire tumor was composed of cartilage and bone in various stages of maturation (Fig. 8). Masses of atypical cartilaginous tissue were seen shading into bone formation, resulting frequently in

an irregular bony periphery surrounding a central island of cartilaginous tissue. The osseous tissue took the form of branching and discrete bony spicules lined by a fibrous layer. These spicules made their way quite freely through the surrounding muscle tissue. One of the striking features was the lack of reaction shown by the surrounding tissue; apart from very slight atrophy of the immediately adjacent muscle fibers, little or no changes were seen in the face of the advancing bony spicules, and, occasionally, longitudinal muscle fibers were seen shading directly into bone. In the subcutaneous regions, spicules of bone invaded the loose areolar tissue with only slight amounts of fibrous periosteum surrounding the osseous portions. In general, there was little in the sections to indicate that the growth was a true tumor.

Diagnosis.—Gross and microscopic findings pointed strongly to progressive myositis ossificans.

Subsequent Course.—One year after amputation, the patient has shown no sign of recurrence, either in the stump or elsewhere, and her general condition is perfectly satisfactory.

COMMENT

Confronted by this clinical and pathologic picture, one cannot fail to be impressed by the distinctly dual characteristics that the lesion seems to present. To recapitulate briefly, we have a circumscribed tumor showing an outer bone shell arising from the head of the fibula and allowing the peroneal nerve to traverse it in a deep groove. Subjected to a microscopic pathologic study, it presents many of the features of a sclerosing osteogenic sarcoma or ossifying fibroma of questionable malignancy, logically interpreted as tumor tissue regardless of its exact subclassification. Following the removal of this tumor, recurrence was first noticed fourteen months after operation, with subsequent rapid growth necessitating amputation of the leg. This time the roentgenologic and the pathologic picture were quite different, and the lesion apparently appeared as a progressive myositis ossificans.

In the light of our present knowledge, an attempt to explain this rather unusual series of events is somewhat hazardous. If, however, a discussion of the diagnostic possibilities is the proper method of attack, we find that four major possibilities seem to present themselves for consideration.

The first possibility is the suggestion that we are dealing with two distinct and unrelated lesions. The supposition is, that on the surgical removal of a sclerosing osteogenic sarcoma, progressive ossification of muscles in and about the operative site develops purely as a result of the operation. This seems indeed to be a remote consideration. Following bone operations, ossification of muscles and surrounding tissues may occur, possibly as the result of trauma, hemorrhage or periosteal displacement, but the occurrence of progressive ossification similar to that seen in this case is indeed unusual. Furthermore, it seems unlikely that there should be no etiologic relationship between these two lesions,

for, although on superficial comparison they may appear as essentially different processes, there are several characteristics common to both.

The second possibility is that we are dealing with one distinct lesion, and that the whole process is in reality that of a progressive myositis ossificans, erroneously diagnosed at the time of the first operation. A study of the initial growth, however, seems to make this view unlikely. Unquestionably, some of its features are imitated by the recurrence, but, in general, the characteristics of the first growth include those of a tumor, and the growth should be interpreted as such.

The third possibility, like the second, suggests that we are dealing with two different aspects of one and the same lesion, namely, a slow growing, sclerosing osteogenic sarcoma, which recurs locally, after operation in an extremely atypical form bearing, as it does, a striking resemblance to myositis ossificans. In other words, the process is essentially neoplastic and the picture is confused by the atypical characteristics of the recurrence, whose appearance would seem to have been transformed by surgical intervention. This view has many points in its favor. The very fact that the lesion recurred in the operative site may suggest a tumor. The progressive, active growth of new bone, which, besides involving muscle and fascia, also seems to infiltrate the subcutaneous fat, may further emphasize a neoplastic type of lesion. However, although the question of assigning this case to an exact classification may seem to be an academic one, I do not feel that, in listing this process as a primary sclerosing osteogenic sarcoma with an atypical recurrence, one is employing sufficiently comprehensive terms.

Finally, the fourth possibility, which suggests itself as an explanation of this process, is that in the first lesion we are perhaps dealing with two processes standing in distinct relationship to each other, and in the second a postoperative recurrence of one of them. We have a primary osteogenic sarcoma developing in a person who possesses a diathesis for bone formation.[1] The suggestion implies that the actual presence of this initial neoplastic lesion may have served as a stimulus for the development of previously latent, osteogenic change within the muscles and surrounding tissues. Analogy may perhaps be drawn in the examples of traumatic myositis ossificans, complicating healed fractures and callous formations. Certainly, the peripheral characteristics of the primary growth in this case are highly suggestive of this type of process, and are imitated by the picture seen in recurrence. In particular, one finds in this outer bony shell, a distinct similarity in both gross and microscopic appearance to the entire recurring lesion. Furthermore, the manner in which the advancing edge of the first

1. Painter, C. F.: A Consideration of the Etiologic Factors in Myositis Ossificans Traumatica, Boston M. & S. J. **185**:45 (July) 1921.

growth seems to encapsulate, rather than to invade the shaft of the adjacent tibia and fibula, is again evident in the recurrence; in both instances, there is an apparent consideration shown by the growth of bone to certain structures other than muscle tissue, which include the common peroneal nerve in the first instance and the larger arteries of the leg in the second. Probably the tumor was eradicated by the first operation, but at the same time the surrounding tissues may well have been subjected to trauma, hemorrhage and possible periosteal displacement. Subsequent to the operation, recurrence occurs, not of the neoplasm but of the associated ossification of surrounding muscles.

This final interpretation has distinct theoretical aspects, but it has been adopted as being the most satisfactory in the light of our present knowledge of this case. As is often the case in questionable bone tumors, various opinions are given by different pathologists. This case is no exception to the rule, and not all the men who have kindly assisted me in this study have accepted the foregoing explanation.

FURTHER OBSERVATIONS ON THE SURVIVAL OF BONE AFTER REMOVAL FROM THE BODY*

S. L. HAAS, M.D.

SAN FRANCISCO

Somatic death or death of the individual takes place immediately on the permanent failure to function of any one of the three indispensable organs, the integrity of which is essential to life; namely, the organs of the respiratory, the circulatory or the nervous system.

Molecular death or death of the tissues may not ensue immediately, because certain tissues are able to remain viable for variable lengths of time, on the nutritive material stored within the cells or surrounding them. The period of survival is also dependent on the resistance of the cells, the readiness of bacterial invasion and the presence of destructive enzymes. It may be possible, by several methods, to prolong further the life properties of those tissues which are able to survive.

By the first method, so well exemplified in tissue culture, the necessary nutritive materials may be artificially supplied. It has thus been possible to keep a strain of connective tissue alive for over ten years.

The second method of prolonging the life properties of tissue is subjection to low temperatures, whereby the metabolic activities and destructive agencies are suppressed.

As the present investigation is concerned with the behavior of bone when subjected to low temperatures, it may be well to draw attention to a few biologic principles concerning the effects of temperature on life phenomena. In the first place, temperature influences living matter in two ways; namely, chemically and physically. Thus, from the chemical standpoint, the velocity of the chemical reactions of the cells may be retarded or accelerated; and from the physical standpoint, the liquid constituents of the cells may have their state so changed as to pass from the fluid to the coagulable state.

The extreme upper limit of life for high temperatures would be at that point at which the coagulation of the protein of the cell takes place. This varies with the differences in the amount of water the cells contain and also with the reaction of the mediums, because the coagulation point is higher in alkaline and lower in acid mediums. A temperature of 45 C. is generally considered fatal for warm blooded animals

* From the Surgical Laboratory of Stanford Medical School.

within a few minutes. Certain algae will stand a temperature as high as 73 C., and it is well known that some of the spores of bacteria will resist steam under pressure for a number of hours.

At low temperatures, the rate of chemical reaction becomes slower as the temperature is reduced, so that, at 0 C., they become so weak that the manifestations of life practically cease. The lack of water exerts a similar affect on the cells, a fact that is of great importance to the retained life properties of seeds. It is claimed that there is no temperature so low that amebas and some other organisms cannot withstand. It is also claimed that the frog and other cold blooded animals can be brought back to life after being frozen, provided the thawing out is not carried on too rapidly. If ice crystals form in cells that are frozen, the cells may be killed by the mechanical injury which is caused by the crystals.

It is remarkable what extreme ranges of temperature some of the lower forms of life are able to stand. Certain forms of bacterial spores will resist the temperature of steam under pressure; while at the other end of the scale, as has been shown by MacFadyen,[1] various pathologic bacteria will retain their vitality after immersion in liquid air at —190 C. for six months; and Dewar[2] obtained like results with bacteria subjected to boiling liquid hydrogen near —250 C.

Higher in the animal kingdom, there is not this same resistance to temperature, as it has been found that in warm blooded animals, if the blood is cooled below 15 C. for some time, the animal cannot recover. It is fortunate for those animals that are exposed to temperatures below this point that a protective layer of warm air forms on the surface of the body, thereby insulating the cells of the body from immediate contact with the surrounding air.

Even though a warm blooded animal dies if its blood is cooled below 15 C., the tissues of that animal will, more than likely, resist a much lower temperature. It would be expected that bone, with its high mineral content and its freedom from destructive agencies, would be quite prone to withstand a low temperature. On the other hand, a highly specialized tissue, such as brain, would be much more amenable to destruction when exposed to freezing temperatures. The question arises as to the limit of reduction that a particular tissue will withstand. Even though it is admitted that certain forms of animal life will resist any degree of low temperature, it is more than probable that the individual tissues of the warm blooded animals will show a relative sensitiveness, as does the host, and will succumb at a temperature somewhat near the death point of the animal. It may also be asked whether

1. MacFadyen: Proc. Roy. Soc., Oct. 31, 1902.
2. Dewar: Quoted from Adami, General Pathology **1**, 1908-1911.

there is a limit of time that tissues will survive when exposed to low temperatures? In a previous investigation,[3] it was found that bone showed no signs of proliferation after exposure to air for about twenty-four hours. It would be expected that at a lower temperature, life would be prolonged; although it is believed that there would be a limit for the persistence of such living properties. It is interesting to note that Lambert[4] found that embryonic chicken and rat tissue preserved at low temperatures ranging from — 7 to + 20 C. lived longest at + 6 C. The duration of life under the most favorable conditions was less than twenty days.

Several methods for determining the life properties of cells are utilized. The mere microscopic appearance of the nucleus is unreliable, because a cell may die before there are demonstrable changes in the nucleus. The signs of growth, as found for some tissues on culture mediums, have thus far not been conclusively proved for bone. In a previous article,[5] it was shown that there was a definite response of the live osteogenetic cells in the formation of callus, when a transplanted fractured bone is buried in muscle; and it is believed that this physiologic response offers a satisfactory criterion for the presence of life in osseous tissue.

The exact method utilized in the present investigation is as follows: Under full ether anesthesia and with the usual aseptic technic, two metacarpal bones were removed from a dog's foot. Each bone was completely fractured in the middle and then placed in separate sterile bottles. One bone was then placed in the refrigator at about freezing temperature and the other kept at room temperature. At the end of a predetermined time, the fragments of the bone that was kept at freezing point were bound together in normal apposition and buried in the muscles of the back of the same dog from which they were originally removed. The fragments that were kept at room temperature, after being placed in normal apposition were buried in the muscles on the opposite side of the back. After about five weeks, the bones were removed and a microscopic study was made, particularly of the site of fracture, for signs of new bone or cartilage.

EXPERIMENTAL DATA

EXPERIMENT 1.—*Bone kept at freezing temperature for two days. Method.*— The third right metacarpal bone was removed from Dog 37, a growing animal,

3. Haas, S. L.: A Study of the Viability of Bone After Removal from the Body, Arch. Surg. **7**:213 (July) 1923.

4. Lambert, R. A.: The Influence of Temperature and Fluid Medium on the Survival of Embryonic Tissues in Vitro, J. Exper. Med. **18**:406, 1913.

5. Haas, S. L.: Spontaneous Healing Inherent in Transplanted Bone, J. Bone & Joint Surg. **4**:209-214 (April) 1922.

and fractured completely at the middle. The two fragments were placed in a sterile bottle and kept in the refrigerator for two days, at a temperature near the freezing point. The fragments were then placed in end to end apposition and buried in the muscles on the left side of the spine of the original animal, from which place they were removed at the end of thirty-four days.

Gross Findings.—The fragments were found in good alinement and appeared to be partially united. Figure 1 shows some callus about the site of the fracture.

Microscopic Findings.—There was a projecting mass of osseous callus extending from one fragment toward the other. At the extreme tip of this new bone, there was a knob of callus (Fig. 2). There was also a considerable outgrowth of callus from the other fragment, from both the periosteal and the endosteal surfaces. There was a definite attempt at union, which appeared

Fig. 1 (Experiment 1).—A fractured bone that was kept at freezing temperature for two days, after which it was placed in the muscles of the back of the animal from which it was originally removed. A small amount of callus can be seen at the site of fracture.

from the sections to be incomplete. A considerable number of the nuclei about the haversian canals were well stained.

EXPERIMENT 2.—*Control bone kept at room temperature for two days. Method.*—The fourth right metacarpal was removed from the same dog at the same time as the bone in Experiment 1. This bone, after being fractured, was kept at room temperature for two days, after which the apposed fragments were buried in the muscles of the back from which place they were removed at the end of thirty-four days.

Gross Findnigs.—There was absolutely no sign of union, the fractured ends were pointed and the bone appeared dead. Figure 3 shows the nonunion and the atrophy of the bone.

Microscopic Findings.—There was no sign of proliferation at any place; in fact, the nuclei were practically all stainless.

Comparison.—After exposure for two days at a freezing temperature, there were almost a sufficient number of live osteogenetic cells to form a union of the bone after being buried in the muscles of the back. On the other hand, the bone that was exposed to the air and similarly treated showed no signs of life and had all the appearance of being dead.

EXPERIMENT 3.—*Bone kept at freezing temperature for three days. Method.*—The third right metacarpal bone was removed from Dog 39, a full grown

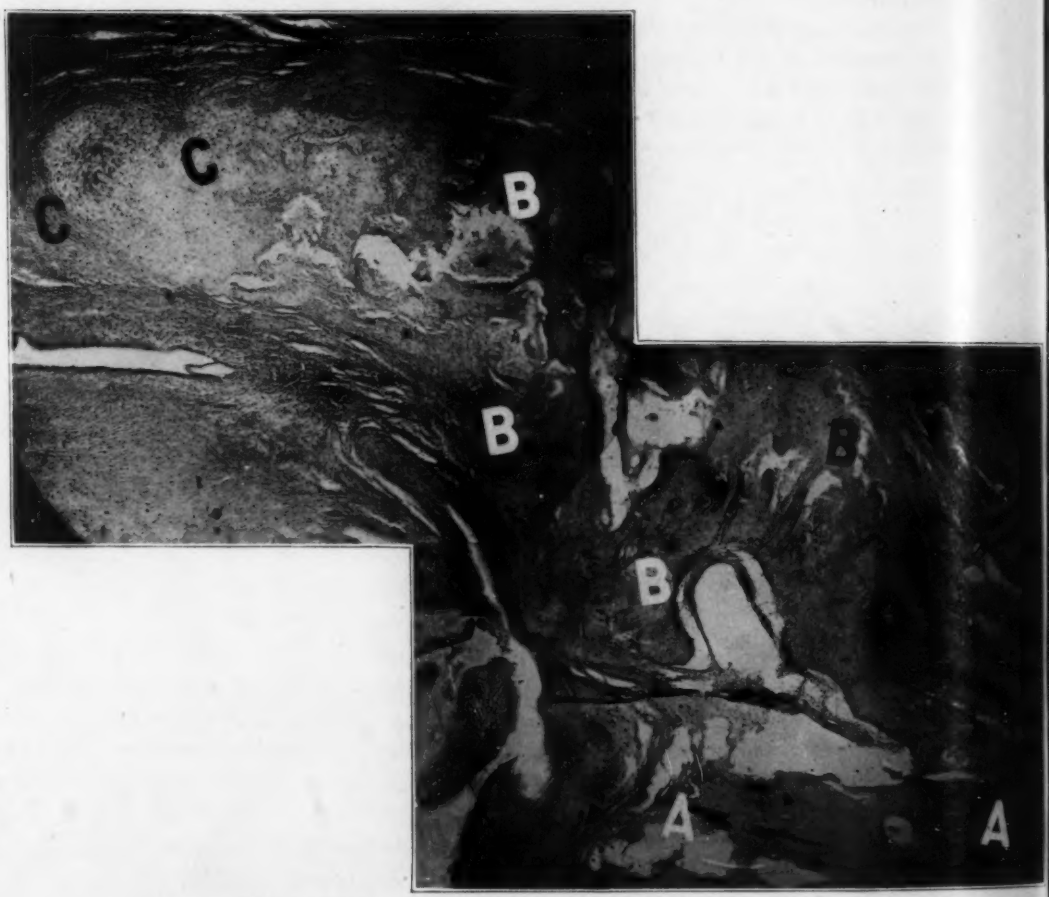

Fig. 2 (Experiment 1).—Bone kept at freezing temperature for two days. Callus formed on one fragment of a fractured bone that was kept at a freezing temperature for two days, and then buried in muscle of the same animal. The specimen shown was removed at the end of thirty-four days; *A*, original bone with stainless nuclei; *B*, new osteoid callus at the extreme tip of which there is a mass of cartilage, *C*.

animal. The bone was fractured in the middle and the fragments placed and kept in a sterile bottle at a freezing temperature for three days. The apposed fragments were then transplanted to the muscles of the back of the same animal, from which they were removed at the end of thirty-one days.

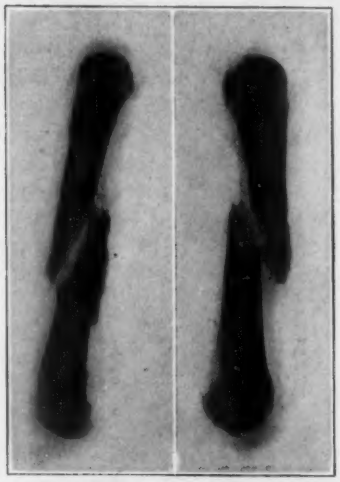

Fig. 3 (Experiment 2).—A fractured bone that was kept at room temperature for two days, after which it was placed in the muscles of the back and allowed to remain for thirty-four days. There was no sign of union or callus. There was considerable absorption of the bone. (Compare with Fig. 1.)

Fig. 4 (Experiment 3).—A fractured bone that was allowed to remain at freezing temperature for three days and then buried in the muscles of the back. There was callus at the site of fracture, which was not so evident in the roentgenogram as on microscopic examination because of the lack of calcification.

Gross Findings.—The fragments were found in good alinement, and there was some stability at the site of fracture. Figure 4 shows practically no absorption, and there appears to be some callus at the fracture site.

Microscopic Findings.—There was a considerable amount of osteoid callus between the two fragments which tended to bridge the gap between them (Fig. 5). The greater proportion of the nuclei of the cortical bone was stainless.

EXPERIMENT 4.—*Control bone kept at room temperature for three days. Method.*—The fourth metacarpal bone was removed from Dog 39 and similarly prepared and treated as the bone in Experiment 3, except that it was kept at room temperature for three days.

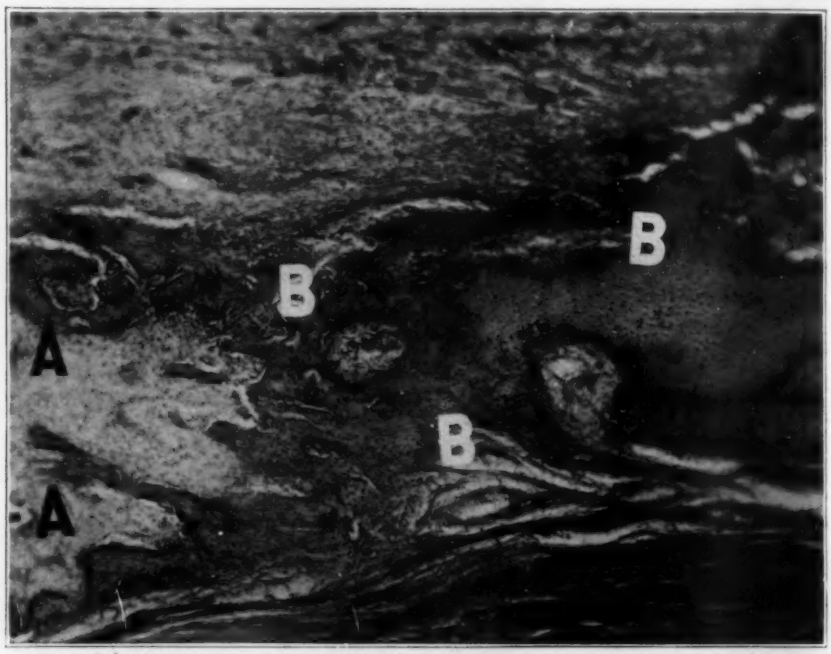

Fig. 5 (Experiment 3).—Callus formed between the fragments of a fractured bone that were kept at a freezing temperature for three days, after which they were placed in a muscle of the back. The specimen shown was removed in thirty-one days; *A*, original bone showing stainless nuclei; *B*, new osteoid callus extending over to the other fragment.

Gross Findings.—There was no sign of proliferation or attempt at union. There was considerable absorption and the bone appeared dead. Figure 6 shows this condition.

Microscopic Findings.—There was no evidence of new bone or signs of proliferation at any place. The nuclei of the cortex were stainless. The end of the bone was blunt and was distinctly demarcated from the surrounding tissue (Fig. 7).

Comparison.—There was a very marked difference between the bone that was kept at freezing temperature and the bone at room temperature. The

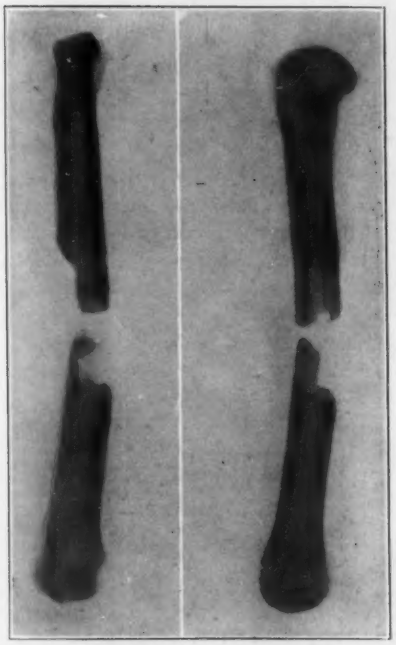

Fig. 6 (Experiment 4).—A fractured bone that was kept at room temperature for three days, after which the apposed fragments were placed in the muscles of the back for thirty-one days. There was no sign of union and the bones showed absorption. (Compare with Fig. 4.)

Fig. 7 (Experiment 4).—This bone, which was kept at room temperature for three days, showed no evidence of proliferation at any place; while the bone shown in Figure 5, which was similarly treated except that it was kept at freezing temperature for three days, showed marked formation of callus. (Compare Figs. 5 and 7).

latter showed a complete death and failure of osteogenesis, while the bone that was kept at the low temperature showed a very active reparative property.

EXPERIMENT 5.—*Bone kept at freezing temperature for five days. Method.*—The third metacarpal bone was removed, and fractured at the middle; and the fragments were placed in a sterile bottle and kept at freezing temperature. At the end of five days, the apposed segments were buried in the muscles on the left side of the back, from which they were removed at the end of thirty-eight days.

Gross Findings.—The fragments were in good alinement, and there appeared to be a soft union. There was considerable absorption and the cortical bone was white.

Microscopic Findings.—There was a small nodule of new bone just back of the fracture plane on both fragments (Figs. 8 and 9). There was no callus

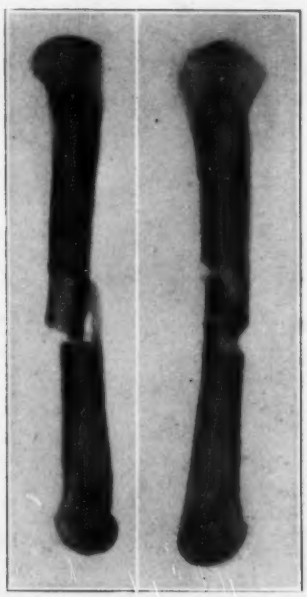

Fig. 8 (Experiment 5).—A fractured bone that was kept at freezing temperature for five days, after which it was buried in the muscles of the back of the original animal. There was only a slight suggestion of proliferation at the site of fracture; in fact, it could hardly be distinguished from the bone that was exposed to the air for the same time. Microscopically, there was some definite cartilaginous and osteoid callus, as shown in Figures 9 and 10.

connecting the two fragments. A considerable amount of new bone filled one of the suture holes that was bored in the bone at the time of the operation. There were a considerable number of stainable nuclei on the periosteal surface and in some places marked endosteal proliferation.

EXPERIMENT 6.—*Control bone kept at room temperature for five days. Method.*—The fourth metacarpal bone from the animal used in Experiment 5 was similarly treated, with the exception that it was kept at room temperature and placed on the opposite side of the back.

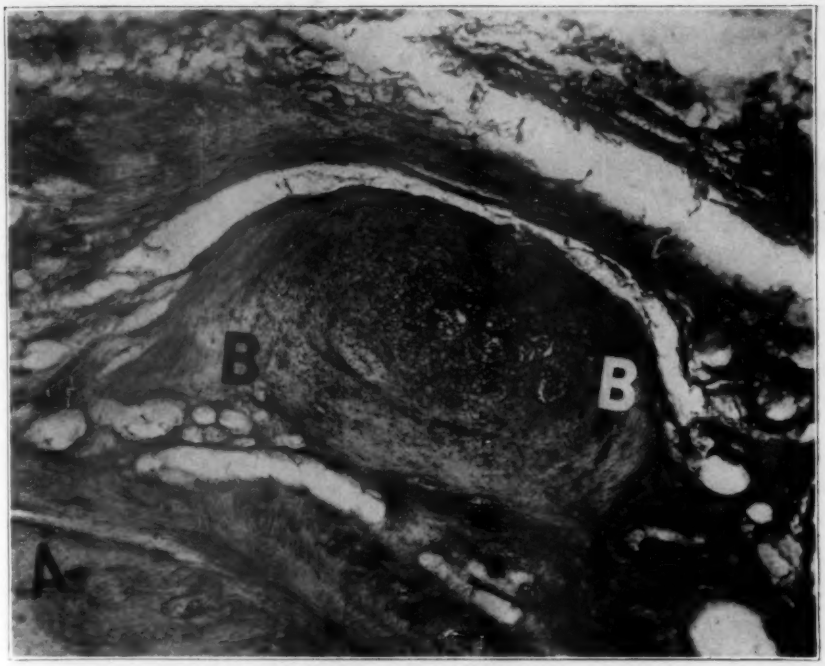

Fig. 9 (Experiment 5).—Cartilaginous callus formed on one fragment of a fractured bone that was kept at freezing temperature for five days, after which the fragments were buried in a muscle of the back of the animal from which they were originally removed; *A*, original bone; *B*, cartilage callus.

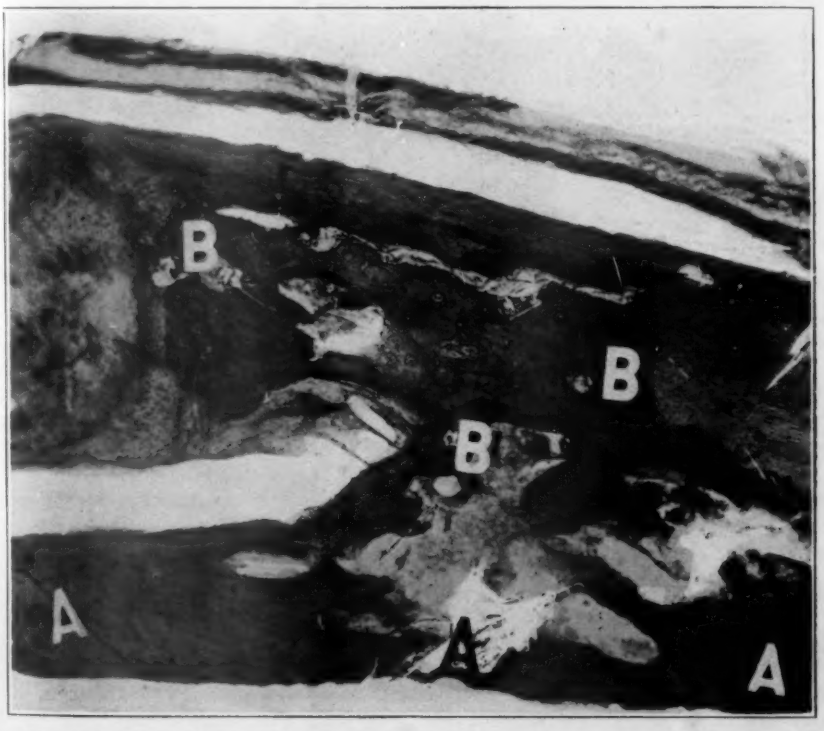

Fig. 10 (Experiment 5).—Osteoid callus formed on the fragment of a fractured bone that was exposed to freezing temperature for five days. This is the opposite fragment to the one shown in Figure 7; *A*, original bone; *B*, osteoid callus.

Gross Findings.—The bone was found embedded in the muscles of the back, with fairly good alinement of the fragments, but no sign of union. The muscle was adherent to the cortex.

Microscopic Findings.—There was a small nodule of bone at one place on the cortex at a considerable distance back from the fracture surface (Fig. 10). This is the only definite new bone that has been found in any experiment in which the exposure period to air has been over one day. Because of the fact that the remainder of the bone appeared dead, it was considered as being an anomaly of metaplastic origin or an outgrowth from cells of retained vitality.

Comparison.—After five days' exposure to freezing temperature, there were definite signs of proliferation about the fractured buried bone. The amount was considerably less than was found after three days exposure, showing that

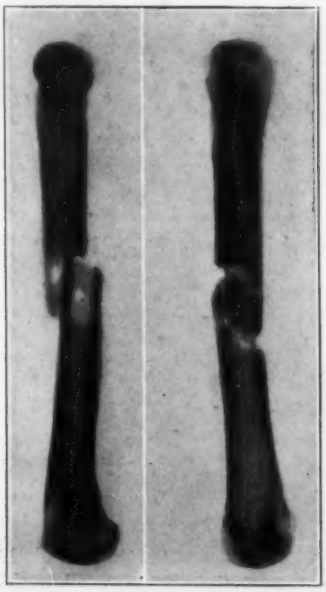

Fig. 11 (Experiment 6).—A fractured bone that was kept at room temperature for five days, after which it was placed in the muscles of the back of the original animal. There was no evidence of proliferation, either in the roentgenogram or microscopically at the site of fracture. A small osteoid mass was found at one place on the cortex at a considerable distance from the fracture surface. (Compare Fig. 18.)

fewer osteogenetic cells remained alive. The bone that was kept in the air for five days showed a small nodule of new osteoid tissue, most likely of metaplastic origin; the remainder of the bone appeared dead.

EXPERIMENT 7.—*Bone kept at freezing temperature for seven days. Method.*—The fourth right metacarpal bone was removed, fractured at the middle, and kept in a sterile bottle at a freezing temperature for seven days. The apposed fragments were then transplanted to the muscles of the left side of the back of the animal from which they were removed, and allowed to remain for nine days.

Gross Findings.—The bone was found lying loose in the muscles, without signs of proliferation.

Microscopic Findings.—The nuclei of the bone stained poorly, and there was a complete lack of any regenerative signs.

EXPERIMENT 8.—*Control bone kept at room temperature for seven days. Method.*—The third metacarpal bone was treated similarly to the fourth of the previous experiment, with the exception that it was kept at room temperature and transplanted to the muscles on the right side of the back.

Gross Findings.—The fragments were found agglutinated to the surrounding muscles without signs of union.

Microscopic Findings.—There was no sign of bone proliferation at any place and the nuclei, with the exception of those near the articular end, were stainless.

Fig. 12 (Experiment 6).—Small amount of new bone found on one fragment of a fractured bone that was exposed to the air for five days before being transplanted into the muscles of the back. This bone was found at a distance from the site of fracture and was either of heteroplastic origin or an outgrowth from some of the cells that had retained their vitality. This was a rare condition, as no other evidence of new bone was found in a number of experiments, in which the time of exposure to air was two days or longer; *A*, original bone; *B*, new bone.

EXPERIMENT 9.—*Bone kept at freezing temperature for seven days. Method.*—The fourth right metacarpal bone was removed from Dog 49, and fractured at the middle, and after being kept at freezing temperature for seven days, the apposed fragments were placed in the muscles on the left side of the spine. The specimen was removed at the end of thirty-seven days.

Gross Findings.—The fragments were found in good alinement, but without any signs of union. The surrounding tissue was adherent to the bone.

Microscopic Findings.—There was no sign of activity about the site of fracture or at any other place on the bone. The nuclei were all stainless.

EXPERIMENT 10.—*Control bone kept at room temperature for seven days. Method.*—The third metacarpal from the same animal was treated similarly to the fourth of Experiment 9, with the exception that it was kept at room temperature.

Gross Findings.—There was no sign of union and the fragments were in poor alinement. The bone was not adherent to the surrounding tissues.

Microscopic Findings.—There was considerable infection of the bone and the nuclei stained poorly.

Comparison.—There was no evidence of proliferation from either the bone that was kept at freezing temperature or that kept at room temperature for seven days.

EXPERIMENT 11.—*Bone kept at freezing temperature for ten days. Method.*—The third right metacarpal bone was removed from Dog 50, a full grown animal; was fractured in the middle, and was placed in a sterile bottle and kept at a freezing temperature for ten days. The fragments were then put in end to end apposition and placed in the muscles on the left side of the back, from which they were removed at the end of twenty-one days.

Gross Findings.—There was no sign of union between the two fragments. The bone appeared fairly normal.

Microscopic Findings.—There was no evidence of proliferation, or were there signs of callus at any place. The nuclei were stainless and the bone appeared dead.

EXPERIMENT 12.—*Control bone kept at room temperature for ten days. Method.*—The same procedure was used as in Experiment 11, except that the bone was kept at room temperature for ten days before being placed in the muscles on the right side of the back.

Gross Findings.—The fragments were found in good alinement but without any sign of union. The bone appeared darker than normal and shelled out from the tissues like a foreign body.

Microscopic Findings.—There was no sign of proliferation and the nuclei were stainless.

Comparison.—There was a complete absence of signs of proliferation after ten days in both the bone that was kept on ice and the one that was kept in the air. The nuclei of the bone in both instances were stainless.

CONCLUSIONS

Utilizing as a criterion the fact that there was a physiologic reparative response of healing of a fractured bone buried in muscle, the following conclusions are presented as a result of this experimental work.

1. There were definite signs of proliferation in fractured bones that were kept at a freezing temperature for as long as five days. The shorter the exposure period, the greater was the reparative response.

2. Control bones that were fractured and kept at room temperature showed no signs of proliferation on exposure for two days or longer.

3. Bone after removal from the body does not die immediately, but retains its vitality for variable periods depending on the environment. The degenerative processes can be retarded, but not completely suppressed, by exposing the bone to a freezing temperature.

4. In order to insure the preservation of the maximum life properties of bone after removal from the body, it is advisable to keep it at a freezing temperature. It is also essential, that such bone be used at an early time, as the vitality of the osteogenetic cells become less as the time increases and is absent after five days.[6]

ABSTRACT OF DISCUSSION

Dr. L. W. Ely, San Francisco: I cannot agree with all the conclusions reached by Dr. Haas. If you cut off any of the tissues of the body from their blood and nerve supply for forty-eight hours, those tissues die. If you put the cut off portion in an icebox, the disintegration of the tissues will be delayed. If you bury this piece after forty-eight hours in the tissue of the animal from which it was taken, it is still dead, and the dead tissue never produces anything. The chief source of error in this study of buried bone is the assumption that dead tissue can produce something. When a piece of bone is buried, two separate and distinct processes go forward in it; on the one hand, distintegration and absorption, and on the other, bone production. According to circumstances, one or the other of these two processes will prevail. If you look at a piece of buried bone, you will see that the bone and marrow are dead, and that new bone and new marrow are being produced. Will you say then that this dead bone and dead marrow produce new bone? No. Everything that happens in that dead piece of bone and marrow, after the time it is buried, is set in motion by the blood vessels pushing into it and starting up new bone production. The mere presence of bone dead or living in the tissues is one stimulus to bone production. If you boil the bone, and bury it in the tissues, after a reasonable length of time you will find that it is being absorbed, but that production is going on. The boiled bone cannot produce anything. The whole process of osteogenesis is started up and maintained by the blood vessels that push in from the outside. If a piece of bone is buried with soft tissues tightly connected with it, that piece will be more apt to persist and be functionally good. That is the reason why so-called live bone is more useful in bone grafting than so-called dead bone. It has a little soft tissue on it, and this soft tissue is quickly vascularized, and the vascularization of the buried bone goes forward more quickly than that of the boiled bone.

Dr. Fred H. Albee, New York: If a detached piece of bone will live, it is the most favorable material we can use to bring about osteogenesis, whether the living bone cells come from the blood vessels of the host tissues, or from the graft itself. As far as practical surgery is concerned, it does not matter. Some years ago, I implanted fresh bone from sheep into dogs for the purpose

6. Since this article was submitted for publication, the work of Grohé has come to my attention. He found that pieces of periosteum that were removed from dead rabbits preserved at 0 C. for as long as 100 hours formed osteoid nodules after being transplanted in muscle (Grohé, B.: Die Vita propria der Zellen des Periost, Virchows Arch. f. path. Anat. u. Physiol. u. f. klin. Med. **5**: 428, 1899).

of ankylosing the vertebrae. The graft produced a remarkable liquefaction as compared with the entire absence of this phenomenon and the immediate primary union of an autogenous graft with the surrounding host tissue. In every instance, the control was accurate. Autogenous implantations (debarring infection) united promptly with the spinous processes, producing a strong ankylosis, whereas the heterogeneous sheep's grafts were not united to the split spinous processes and were found in pockets filled with serum. I saw a case yesterday in which a mutton chop, so-called, had been transplanted for an ununited fracture of the radius. The roentgen ray showed a great cavity in the radius in which was lying what I considered to be the mutton chop. There was no evidence of its having united with the radial fragments. In 1912, I used, in one case, a part of the humerus of an infant strangulated at birth and in the other, part of the femur. Six years after the implantation, both grafts were intact. In 1913, I operated in a case of sarcoma of the tibia in a child, to remove the growth and restore bone by graft. The roentgenogram made before operation apparently indicated that it would be possible to excise the whole malignant area. The history showed that it was of short duration and apparently a favorable case. After a considerable section of the tibia had been removed, it was found impossible to get it outside of the malignant area because the tumor had broken through into the popliteal space. Two grafts, fastened together with kangaroo tendon, were hastily implanted in the bone hiatus, the wound closed and immediate amputation was recommended. The family did not decide to allow amputation until four weeks later, when the leg was amputated. The roentgenogram of that leg showed a union between these two grafts, notwithstanding they were imbedded in the soft parts of the leg, and the callus which had united the graft ends was far distant from any other bone. The diameter of the grafts had not increased in any portion of their extent. There can be no question that in this case the new bone formation uniting the grafts came from the grafts themselves.

Dr. Arthur L. Fisher, San Francisco: In certain portions of Dr. Haas' slides there could be no question but that the tissue which looks like bone could be seen filling in from the outside. The connective tissue shows the changes from ordinary young connective tissue to the osteoid tissue by the so-called process of metaplasia. I think some of that bone unquestionably is formed in that way. It looked to me, in those specimens that I have seen, as if the bone was dead, and the new bone formation had all grown in from the outside.

Dr. S. L. Haas, San Francisco: If you take out a bone, fracture it completely and boil it, then close the apposed fragments in the muscles of the back, there is never a union. No bone is thrown out at any place or near the fracture line. The only evidences of stainable nuclei are found where the ligaments attach. The cells at that particular point are the most persistent cells of the bone. I do not say that they are alive but they retain their staining properties longest. I think that accounts for many of the misinterpretations of living elements in transplanted boiled bone. You never see any signs of bone being thrown out at any place about these buried patellas. In other experiments I boiled one half of a metacarpal bone and left the other half alive. I buried the two fragments in the muscles of the back. There was sufficient osteogenesis in the live half to grow into and form a union with the boiled half. Now I cannot see how anybody can assert that this new bone came from the outside. Furthermore, if the endosteum is scraped out of fracture bones and the fragments are buried in the muscles of the animal's back they will unite in

practically 75 per cent. of the cases. Take another set of fractured bones, scrape off the periosteum and bury them in the muscles of the back. Union occurs in about 75 per cent. of the bones. Remove both the periosteum and the endosteum, and union never occurs, and very rarely will you see any sign of new bone. There you have live buried bone without signs of osteogenesis. When you leave the periosteum in situ osteogenesis occurs; when you leave the endosteum on, osteogenesis also occurs but remove them both, and osteogenesis never occurs. Surely, the removal of the endosteum and periosteum would not prevent the ingrowth of bone from the outside; and these experiments can be interpreted only as showing that the source of osteogenesis must reside in the osteoblasts of the periosteum and endosteum of the transplanted bone. Regarding Dr. Albee's statements, any bone from another animal acts more like a foreign body than a bone from the same individual. In using bone from another individual you have the same chance as in using blood from another person in blood transfusion. I do not want to give the impression that all osteogenesis comes from graft, because it does not. In using a live graft you have osteoblasts tissue thrown out from the graft to the host, as well as invasion from the host into the graft and persistent cells in the graft about the haversian canals—all of which makes a live graft preferable to a dead graft.

SOME OBSERVATIONS ON THE DRAINAGE OF SUBCORTICAL BRAIN ABSCESS *

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The purpose of this paper is to discuss the operative treatment of chronic encapsulated abscess of the brain and to report the results of operation in a small series of abscesses of this type.

In view of the high mortality, any method of operation for brain abscess should be carefully scrutinized to exclude technical errors, which may not only prevent the eradication of a localized suppuration, but actually spread the infection to uninvolved regions of the brain. It is generally appreciated that the principles utilized in the successful treatment of abscess in other regions of the body must be radically modified in operations for brain abscess. An unfailing adherence to these principles in the treatment of the various types of abscess of the brain will often convert a walled-off, more or less quiescent lesion into a rapidly fatal septic encephalitis, the most frequent terminal complication of an unsuccessful operation for brain abscess.

Localized intracranial suppuration occurs in various forms and locations, and it is of advantage to individualize the types of abscess before advocating a plan of treatment that, while effective in one type, may be totally inadequate in another. It is obvious that a collection of pus in the intracranial cavity is a serious menace to the patient's life; but a careful consideration of all the facts justifies the opinion that the gravity of such a situation may be greatly intensified by precipitate efforts at free drainage of the abscess in its early stages, or when it is located at a considerable distance from the cerebral cortex. The best results in the treatment of brain abscess have been obtained in the drainage of single abscesses with a capsule, which constitute the majority of cases coming to operation. If such an abscess is connected with the meninges by adhesions and the evacuation of the pus is properly conducted, the mortality should be very low. It often happens that the abscess is not attached to the meninges, or, if it is, the point of attachment cannot be found. We agree with Eagleton [1] that the so-called stalk of brain abscess is of infrequent formation, and when it does form, the abscess is usually in a late stage. All subcortical abscesses pass through an initial stage of localized encephalitis, followed

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* Read before the Southern Surgical Association, White Sulphur Springs, Va., Dec. 12, 1923.

1. Eagleton, W. P.: Brain Abscess, New York, the Macmillan Company, 1922.

by pus formation and, in most cases, by the formation of a capsule. It is hardly probable that operative treatment would be helpful in the stage of acute diffuse encephalitis, nor does free exploration of a nonencapsulated collection of pus below the cortex appear to offer the patient good prospects for recovery.

In a general way, there are two recognized methods of dealing with a subcortical abscess. Eagleton [1] appears to favor a wide exposure in all cases because of the possibility of a meningeal abscess, which would be overlooked with a small bone opening and which cannot be differentiated from a subcortical abscess prior to exploration. A number of surgeons prefer a small cranial opening and the immediate or delayed insertion of a small rubber catheter into the abscess cavity after it has been located with a ventricular needle. There are obvious objections to an osteoplastic flap for brain abscess. Flap operations are procedures of considerable magnitude in very ill patients, and if pus is evacuated, the bone almost always becomes infected, a troublesome chronic osteomyelitis resulting. The argument for a flap might be disposed of by a preliminary puncture through a trephine opening in the line of incision for the proposed flap, and if pus is not found, the flap can be raised; whereas, if an abscess is located, the small opening is safer and as effective as the flap for draining the abscess. Both the advocates of the bone flap and of simple exploration through a small cranial perforation finally drain the abscess through a short dural incision, so that the extensive procedure of raising the flap is entirely unnecessary in the actual evacuation of a subcortical abscess. In two cases of abscess, we used the osteoplastic flap because we could not be sure before operation whether we were dealing with a tumor or an abscess. In each case, the patient had extensive osteomyelitis, with recovery after a number of months.

The critical stage in the treatment of brain abscess is the time of the discovery of pus by the exploring needle. It is sometimes difficult to determine just how drainage of the pus should be accomplished, or whether aspiration with a needle alone should be made. It is highly probable that, in many cases, the drainage tube does not enter the abscess cavity but lies in the brain tissues about the abscess, traumatizing the unprotected brain and causing septic encephalitis. In deep-seated small abscesses, the instrument used for exploration, regardless of its type, should not be withdrawn, because it is almost impossible to reenter such an abscess with any form of drainage tube. To avoid losing the abscess by collapse of the sac, we have fitted the ventricular needle with a cannula which may be pushed over the needle into the abscess cavity before the needle is withdrawn. This cannula may be used for drainage until the drainage canal is well formed, when the eye end of a small rubber catheter may be substituted.

Dowman [2] has recently advocated the aspiration of a small amount of pus from the abscess and the insertion of a strip of folded rubber tissue down to, but not into, the abscess. Later on, the tissue is withdrawn and a rubber catheter is pushed into the abscess cavity, which has been carefully localized. The advantage claimed for this method of graduated drainage is the formation of adhesions that exclude the general subarachnoid space from infection by the pus from the abscess. In the cases of subcortical abscess that we have drained, the cortex did not lose its firm contact with the dura, although as much as 5 ounces (148 c.c.) of pus was evacuated in one case.

Contamination of the meningeal spaces has not been dreaded so much as a septic encephalitis, which is likely to follow any fumbling with the primary drainage. In some of the cases considered in this discussion it would have been impossible, because of the tension, to have prevented the escape of pus after withdrawal of the needle, even with an abscess 3 cm. below the cortex, and in these cases the Dowman method would have failed of its purpose. When a chronic encapsulated abscess is located with a ventricular needle, suitable drainage should immediately be instituted. There is no other time so favorable for placing a drain in such an abscess cavity as when pus is first discovered. Brain abscess sacs collapse after partial evacuation, and efforts to reenter them often fail. The result is too frequently a traumatic encephalitis. It is surprising how completely an abscess will evacuate its contents under favorable conditions at the primary operation, with little subsequent discharge; which is due largely to the process of repair and to reaction to the drainage material. In three cases, the evacuation of a large amount of pus at the primary operation was followed by discharge of a considerable quantity of clear fluid. This occasioned some apprehension in the first case for fear the ventricle had been entered. The source of the fluid is probably the edema of the brain tissues, and it is helpful in irrigating the abscess cavity. If the abscess cavity is within approximately 4 cm. of the surface of the cortex and contains as much as 30 c.c. of pus, a small rubber catheter lubricated with petrolatum is substituted for the cannula as soon as the pus has ceased to flow freely. This catheter is carefully sutured to the scalp and is not removed until drainage is permanently discontinued. We learned in the first two cases that drainage from a brain abscess is not interfered with by obstruction of the tubes. Sudden cessation of drainage with an increase of existing symptoms, or an accession of new symptoms, generally means a complication arising from no fault of the tube itself.

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2. Dowman, C. E.: The Treatment of Brain Abscess by the Induction of Protective Adhesions Between the Brain Cortex and the Dura Before the Establishment of Drainage, Arch. Surg. **6**:747 (May) 1923.

The importance of the effects of gravity has not been properly appreciated or utilized in the treatment of brain abscess, as has been done in lung abscess. The satisfactory results of operation on encapsulated temporal lobe abscess when the abscess cavity is entered at its lowest level illustrates this point. If during operation for abscess in any location, the patient's head is lowered or turned so that the drainage tube is more dependent, the escape of pus is often greatly facilitated.

One of the most dreaded complications of abscess operations is fungus of the brain, but we do not agree with those who rate it as one of the prime causes of the high mortality. When a fungus develops, as it has usually done in my experience when the abscess was drained through the vault, it generally means that the patient has an extension of the encephalitis, an accumulation of pus not reached by the drainage tube or obstructive hydrocephalus. A dural incision not longer than 1.5 cm. and partly filled by a tube is large enough to permit an extrusion of brain tissue. We have not felt that a shorter incision could be satisfactorily employed if the cortical veins are to be avoided with certainty. If a fungus develops, the shorter the dural opening, the larger the fungus may become, from mechanical reasons. A fungus, large or small, developed in four of our seven cases of subcortical abscess attended with recovery. In two cases, the fungus was of great size, and in one fatal case, the patient had a double fungus. This patient, on admission, had a fungus through a decompression opening which had been made in another clinic. The second fungus in this case followed drainage of one of three abscesses through a burr opening in the midfrontal region. The complication of a fungus greatly prolongs the patient's stay and adds generally to the postoperative difficulties. Protection of the fungus by rubber tissue and a doughnut pad, with daily spinal punctures, if the intracranial pressure is raised, seems to be the best treatment. When the pressure determined at lumbar puncture is about normal, a large mushroom like fungus protruding through a small dural opening may be removed with a cautery, but operation on the fungus is dangerous and futile if the intracranial pressure is high. One patient with fungus was given a subtemporal decompression, followed by cautery amputation of the extruded brain and daily spinal punctures. This treatment was very satisfactory.

No effort has been made to inspect the interior of an abscess cavity or to cleanse the abscess with irrigation. Either of these procedures would be feasible when the cortex is attached to the dura, but either would seem inadvisable when the abscess is subcortical and there is no fusion of the cortex with the overlying dura.

The views expressed in this paper are based largely on a series of ten encapsulated subcortical abscesses, with three deaths. Operation for drainage of the abscesses was performed on nine patients.

Simple exploration with drainage through a small trephine opening was used in seven cases, and a bone flap was made in two cases because of an uncertain preoperative diagnosis. In these two cases, the flap operation was of no special advantage because the abscess was located at the first puncture of the ventricular needle and was drained through a short dural incision, which could have been made just as well through a small trephine opening in the bone.

One patient died during the first visit, without operation, from general meningitis following a ruptured left frontal abscess, discovered at necropsy. A second patient was operated on after the onset of meningitis and a ruptured abscess of the left temporal lobe was found and drained. Purulent spinal fluid was removed at lumbar puncture before operation. The third fatal case was that of a patient with three right frontal abscesses, who was admitted to the hospital with a large temporal fungus through a decompression opening, for the relief of general pressure. In this case, one abscess was localized and drained, but two were overlooked. The fungus could not be controlled, and the patient died from septic encephalitis about six weeks after coming under our observation. Seven of the nine patients undergoing operation recovered. One patient is still in the hospital, but seems to be free from abscess symptoms. She had an enormous fungus cerebri when admitted to the hospital, following drainage of a right parietal lobe abscess, from osteomyelitis of the skull. Two additional abscesses were drained.

Another patient who was discharged from the hospital three months ago has left hemiplegia, involving chiefly the arm. One patient has a slight osteomyelitis of the skull following a flap operation. Still another patient, with left frontal abscess, recovered about a year ago from abscess symptoms, but is troubled with slight headache. There are no other residuals in the series.

A detailed report of these cases will be published in a subsequent paper.

TWENTY-FIFTH REPORT OF PROGRESS IN ORTHOPEDIC SURGERY *

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CONGENITAL DEFORMITIES

Meyer,[1] after studying the roentgenograms of all his patients with congenital dislocation of the hip in regard to the amount of antetorsion of the femoral neck, and the frequency of reluxation, and after measuring by a special scale all the infantile hips at the Berlin Orthopedic Clinic, comes to the conclusion that the influence of antetorsion on the reluxation has been generally overestimated.

Dickson [2] reports the results of operation in cases of old congenital dislocation of the hip. He says that before deciding on an operation very careful attention must be given to the extent to which this condition handicaps the patient. If the general endurance and capacity for work is seriously interfered with, and if the operation can give a stable hip without any considerable limitation of action and definite incapacity in the amount of shortening of the leg, Dickson feels that the operation is justifiable. In four of his five cases, he obtained successful results, and in one a stiff joint due to infection. The operation consists in forming a new acetabulum by turning down a flap from the ilium after the manner of Albee. The successful results were due to stabilization more than to any increase in length of leg.

[ED. NOTE.—We agree with Dickson in what he says about the necessity of careful attention to the extent of the handicap of the patient caused by the old dislocation. We would even go further and try out supportive methods and rest, which, in many cases, when carefully carried out, have relieved all symptoms.]

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* This report of progress is based on a review of 346 articles selected from 620 titles dealing with orthopedic surgery, appearing in medical literature, between April 25, 1924, and Aug. 9, 1924. Only those papers which seem to represent progress have been selected for note and comment.

1. Meyer, Albrecht: Arch. f. Orth. u. Unfall-Chir. **22**:240, 1923.
2. Dickson, F. D.: J. Bone and Joint Surg. **6**:262 (April) 1924.

Ludloff has seen an actual transformation of a clubfoot into a flatfoot by nothing but a dissection of all insertions of the posterior tibial tendon insertions at the tarsal and metatarsal bones. On the other hand, a flatfoot will be transformed into a moderately hollowed foot with even slight supination by putting the short personeal into the tendon sheath of the weak posterior tibial and fastening it at the medial insertion of that muscle. Ludloff promises further studies on this question.

Davenport [3] and his co-workers present an excellent work on radio-ulnar synostosis. The condition is dealt with from the developmental and hereditary point of view. They feel that the condition is a dominant, partially sex limited trait which varies in degree of expression.

TUBERCULOSIS

Prevertebral Tubercular Abscesses.—Valtancoli [4] reports on the method and the experiences at the Rizzoli Institute at Bologna of the treatment of prevertebral tubercular abscesses in the middorsal region complicated by paraplegia. The seriousness of the lesion is shown by the fact that thirty of the thirty-nine cases of paraplegia occurring in 1,004 cases of spondylitis occurred in lesions of the middorsal region. The seriousness of paraplegia is further shown by the fact that while the mortality in their series of spondylitis was 16.3 per cent., in the cases with paraplegia it was 43.2 per cent.

Various methods have been devised to evacuate the abscess. Among them, that of Professor Menard, who performed in twenty-four cases a costotransversectomy, consisting of the resection of the cervical portion of the rib and of the corresponding transverse process. This method has been abandoned on account of the fistula which results. Calvé recently devised a method of inserting a special instrument through the intervertebral foramen and thus evacuated the abscess from within the spinal canal. The method in use at the Rizzoli Institute has been devised after careful anatomic and roentgenologic studies, and consists in inserting a trocar of a diameter of not less than 3 mm. through the intercostal space, starting at a point about two fingers breadth from the spinous process and proceeding in a direction from without inward, following the margin of the body of the vertebra, and striving to keep in contact with the superior margin of the rib below. As a rule, the puncture is made at the right of the spine, as there is less danger of puncturing the aorta even if it is embedded in the wall of the abscess. The depth of the abscess from the skin is about 7 cm. To date, the

3. Davenport, C. B.; Taylor, H. L., and Nelson, L. A.: Radio-Ulnar Synostosis, Arch. Surg. **8**:705 (May) 1924.

4. Valtancoli, Giovanni: Chir. d. org. di movimento **8**:497 (June) 1924.

method has been used on eight patients. In four of these, paraplegia was present. Of these four, in three a notable quantity of pus was expressed; in one, none. In the three cases with evacuation of pus, there occurred complete restoration of the function of the legs in one case, a marked improvement in a second case, and no change in the third. In the last case, the paraplegia had existed for about one year. On account of the encouraging results obtained, it has been used in four cases in which there were large abscesses as a measure to prevent paraplegia. His conclusions are:

1. In every case of paraplegia due to tuberculous spondylitis, in which the roentgenogram shows the presence of a mediastinal abscess which could be reached from without, there is an absolute indication for the evacuation of the abscess.

2. The same strict indication exists also in cases in which, although paraplegia is not present, there are reasons for fearing its appearance, such as subjective weakness of the legs, exaggeration of the tendon reflexes, ankle clonus or phenomenon of Babinski.

Albee's Operation.—Biesalski [5] has studied eight specimens of bone grafts put in after the manner of Albee and taken from spines of children from four months to two years after the time of operation. He was able to remove this material at necropsy, and found that the bone graft does not grow in length. Too early weight-bearing and functional strain may cause pseudarthrosis through the graft. The graft itself cannot correct the deformity, and the disease itself may progress without any influence exerted by the graft.

Association of Pulmonary and Bone and Joint Tuberculosis.—MacKinnon [6] has made a study of thirty cases of adults seen at the Galloway-Gibson Clinic. Tuberculosis in adults is a general disease in which lesions of the lung are almost always present. Bone and joint tuberculosis is not a frequent complication of pulmonary tuberculosis. Evidence of pulmonary infection can usually be found in adults suffering from bone and joint tuberculosis. A large percentage of adults suffering from bone and joint tuberculosis are definitely ill with pulmonary tuberculosis, or are in danger of becoming so, and this fact must be borne in mind in deciding on the treatment of each patient.

Treatment of Surgical Tuberculosis.—Kisch [7] gives a report of the treatment of surgical tuberculosis as it is carried out at the asylum in Hohenlychen, which is connected with Bier's clinic in Berlin. He

5. Biesalski, K.: Arch. f. klin. Chir. **127**:667, 1923.
6. MacKinnon, A. P.: Canad. M. A. J. **14**:124 (Feb.) 1924.
7. Kisch, E.: Deutsche med. Wchnschr. **50**:668 (June 23) 1924.

believes that there is a special advantage in heliotherapy carried out in a flat country as compared with the same treatment carried out in the mountains. Kisch believes that exactly the same results may be obtained by fresh air treatment in winter. There seems to be no reason why patients with pulmonary tuberculosis should not be exposed to the sun's ray, but they must be exposed to it gradually. At the Hohenlychen asylum, the parts are exposed to the sun as much as possible, and during the dark days artificial light is used, but not the usual quartz lamp. Kisch uses one made by Zeiss at Jena. He also uses passive congestion of the type recommended by Bier some years ago, and gives iodid of sodium internally. In addition to this, in some cases the roentgen ray is used. His results are reported as being satisfactory.

Arteries in Surgical Tuberculosis.—Ludloff [8] points out the interesting fact that in three cases in which he performed an amputation of the lower leg for surgical tuberculosis of the foot, the lumen of the arteries was found to be extremely small. There was even no bleeding from the tibial artery. Associated with this was extreme atrophy of the bone. Ludloff believes in an intimate connection between these two findings. From his point of view, such severe atrophy and such a circulatory change presents a prognosis which is very grave.

Periarterial Sympathectomy in Tuberculosis of the Extremities.—Laewen [9] has performed periarterial sympathectomy on nineteen patients who had various types of tuberculosis of the bones or joints of the extremities. Sixteen of these infections were in the leg, and three were in the arm. In four cases, a complete cure was the result. All of them were cases of the severest type, with sinuses of long duration. In five cases, a definite improvement was noted, although Laewen was not at all certain that this might not have followed treatment by rest in bed. In the remaining cases, no improvement was seen. His observations on the extremities of the parts operated on show that the temperature of the skin of the affected extremity rises considerably after periarterial sympathectomy, even in those cases in which it was previously higher than on the sound extremity. He also observes that this rise in temperature is caused by the dilatation of the vessels, and in this he believes lies the chance for beneficial results in the treatment of tuberculosis.

Treatment of Tuberculosis of Bones and Joints.—Girdlestone [10] has written an outstanding article on the modern treatment of tuberculosis of the bones and joints. It is not only outstanding in the breadth of its

8. Ludloff: Zentralbl, f. Chir. **16**:861 (April 19) 1924.
9. Laewen: München med. Wchnschr. No. 7, p. 191, Feb. 15, 1924.
10. Girdlestone, G. R.: Brit. M. J. **1**:1044 (June 12) 1924.

point of view and the soundness of its conclusions, based on a very wide experience and an unusual opportunity to study the results obtained by different methods, but outstanding also in its style and charm of diction, so wofully lacking in much medical literature. He discusses the vast improvement in the management of these conditions during the last quarter of a century, the character of the disease, and its rational treatment. The action of the sun and wind is presented with great clearness, and the importance of both these factors in the essential open air regimen is weighed in a balance so nice as to be convincing and most encouraging to those of us who have no Alps at Leysin on which to care for our patients. He says:

1. The first step in heliotherapy is the carefully graduated exposure of the body in order to develop pigment, and until and unless pigment is developed, only a small dose can be tolerated, and more than a small dose is definitely harmful.

2. Pigmentation, so far as is known, is definitely and merely a protection of the deeper layers of the skin and underlying tissues from the photochemical effect of sunlight. Once the pigment is developed, the pigment metabolism is apparently relatively inactive.

3. Sunlight contains both light and heat; a good skin takes prompt steps to stop the light as near the surface as possible. This is exemplified in the highest degree in the negro. In fact, the better the skin and the more acclimated the race, the more effective a barrier is erected against the photochemical action of sunlight.

4. The better pigmented the patient, the more completely photochemical activity is cut out and the better he responds to heliotherapy. One notices that it is the sun's heat which he is accepting with advantage. On the other hand, I think the photochemical action of sunlight is most valuable in avoiding or correcting a diet or light deficiency. For this the doses needed are small, and there is some evidence that the respiration of air exposed to unfiltered sunlight is enough. Wind and air are heaven-sent factors. Neither can be used to the best advantage without the other. Together they work in ways far beyond direct heliotherapy as a source of health and vigor. They stimulate and cheer us all, and particularly patients in open air wards. Possibly, too, the partnership between sun and air may be more intimate than we know yet. Between the days of white children and these days of brown children there was a decade or so when rosy cheeked children thrived in open air wards.

After having been educated in the Thomas school of "rest, enforced, uninterrupted and prolonged," Girdlestone has observed with great sageness the results at Leysin, under Rollier, and at Alton in England, under Sir Henry Gauvain. At both these hospitals, the patients are immobilized during the active stage, but at Alton as soon as spasm and sensitiveness disappear, they are allowed to move their affected joints in recumbency, most careful observations being constantly made as to return of spasm and pain and more limited movement, a return to com-

plete fixation and traction being demanded. Girdlestone, in spite of his training, inclines to the belief that the latter treatment prevents bone and muscle atrophy and leads to better blood supply. We hope for a wide reading of this sound and stimulating article.

CEREBRAL PALSY (SPASTIC PARALYSIS)

Stoffel's Operation.—Kreuz [11] has reexamined thirty-three of ninety-nine cases in which a partial resection of motor nerves had been carried out, in Gocht's clinic in Berlin. During 1916 and 1917, in half the cases the lengthening of the Achilles tendon was performed in addition to careful resection of the nerves. From 1919, the Achilles tendon has been left intact, and the operation on the nerves has become more radical, the whole supply for the gastrocnemius and the dorsal branch of the soleus having been sectioned, the ventral branch and the branch supplying the flexors of the toes being left intact. Since this more radical nerve operation without lengthening of the Achilles tendon has been done, the recurrences have been less. In several cases in which a secondary operation was performed, extensive neuromas were found. The great importance of careful and persistent after-treatment is emphasized.

Sympathetic Ramisection.—Royle,[12] assisted by Professor Hunter, has continued his investigations on the rôle which the sympathetic innervation of the skeletal muscles plays in the "plastic tone" of these muscles in cases of spasticity. By "plastic tone" they do not mean quite the same thing as Sherrington's postural tonus, but a sort of lead pipe rigidity superimposed on spasticity which makes it more difficult for the patient to move, especially to coordinate the movements of his limbs, and for the observer passively to flex and extend and rotate such a limb. In cases in which this plastic tone exists, a patellar tendon tap, for instance, will result not only in an exaggerated reflex, but also in a tendency for the limb to remain partially extended and not to fall quickly back to its former position. Successive taps may actually result in successive stages of increased extension, until the limb is held for a time almost completely straight. Unless this "plastic tone" exists in a spastic limb, they do not consider, in general, that a case is suitable for the operation of ramisection of the sympathetic fibers, since their theory, seemingly amounting to proof, is that the skeletal muscle fibers have both a medulated and a nonmedulated nerve supply, and that certain fibers are supplied only by the nonmedulated filaments of the sympathetic system. In certain

11. Kreuz: Ztschr. f. orthop. Chir. **45**:356, 1924.
12. Royle, N. D.: M. J. Australia **1**:587 (June 14) 1924.

spastic cases, they think they have established the fact that it is the overaction of these fibers which is responsible for the "plastic tone" which so seriously interferes with muscle control and coordination, and that on the basis of de Boer's[13] experiments, Langelaan [14] is right in maintaining that the ordinary medulated nerves are responsible only for the production of contractile tone. Since Royle's[15] original article, he has modified his operative procedures somewhat after consultation with Professor Hunter, and now removes or avulses only the rami communicantes of the second, third and fourth lumbar ganglions before dividing the main trunk below the fourth lumbar ganglion, thus, in the lumbar operation for the lower limb, avoiding all danger of interfering unintentionally with the function of the intestines or the bladder by leaving the medially directed fibers from the second, third, and fourth lumbar ganglions intact. In the first operation, it was planned to define and divide both the white and gray rami to the second lumbar nerve, in addition to removing the gray rami to the third and fourth lumbar nerves and dividing the abdominal sympathetic trunk below the fourth ganglion. Royle has now performed these cervical and lumbar ramisections many times, both in Australia and in this country, in carefully selected cases, and is still convinced of the great value of the procedure in establishing greater voluntary coordinated control. He believes that the results will be permanent and probably enhanced by time.

[ED. NOTE.—We have had the opportunity of observing these operations performed by Dr. Royle, and have admired the bold conception and precise dissection which has been responsible for their successful accomplishment. The intimate anatomic relation of most vital structures in both the cervical and lumbar operative fields and the many hazards which, if not successfully met, may spell disaster demand of surgeons, first of all, a most diligent perusal of Royle's [12] description of these procedures and, second, most careful preparation on their part by actual cadaver dissection. As we have watched Dr. Royle and Professor Hunter go over a large number of cases which we have been inclined to group under the general name of spastic paralysis, we have been impressed with the comparatively small number of these patients whom they consider suitable for their operation of sympathetic ramisection. As we have previously said, in the Report of Progress, unless the surgeon thoroughly prepares himself to perform these delicate dissections and selects his cases with great intelligence, untoward results

13. De Boer, S.: Ztschr. f. Biol. **65**:239, 1915.
14. Langelaan, J. W.: Brain **38**:235, 1915.
15. Royle, N. D.: M. J. Australia **1**:77 (Jan. 26) 1924.

are sure to follow, and an unjust discredit become attached to a procedure of great promise based on careful experimental work, which, if confirmed, represents a great and far reaching physiologic discovery.]

SURGERY OF THE PERIPHERAL NERVES

The Direct Neurotization of Muscle.—Serra [16] has conducted a series of animal experiments in order to determine whether the possibility of direct neurotization of muscle can be conclusively proved. The results which he reports were studied in a series of twenty-two rabbits. In twenty of the twenty-two in which he made a direct neurotization of the paralyzed gastrocnemius muscle, the results were postive. In the remaining two, definite causes of the failure were established. The experiments were controlled physiologically by electrical stimulations, myograms, neuromuscle preparations and microscopic preparations. The renewal of function in a paralyzed muscle by direct implantation of the central end of a motor nerve, by the implantation of a part of the nerve, and even by employing a free autoplastic transplant uniting the paralyzed muscle with a neighboring healthy nerve, seemed to have been conclusively demonstrated. Comparing the results of these three different methods, it is evident that the implantation of an entire nerve offers the best chance of success. Partial implantation, although it is capable of establishing new control over a paralyzed muscle, presents, nevertheless, the difficulties always present whenever a nerve is sectioned. Neurotization by means of free autoplastic or homoplastic transplants is at least partially successful, as has been demonstrated by the renewal of function and the reappearance of terminal plaques after ninety days. Serra considers that these experiments permit him to state that (1) the method of direct implantation of a motor nerve is efficient in restoring the function of a muscle which has been paralyzed for a considerable period, in one case after a paralysis of 136 days; (2) it is not absolutely necessary to employ an entire nerve; a part of it (roughly one-half) may be sufficient to restore function and prevent trophic disturbances; (3) the same result may be obtained by means of an autoplastic or homoplastic transplant, which may act as a conductor of the nerve stimulus to a muscle from a neighboring healthy nerve; (4) the point of implantation of the nerve into the muscle is of comparatively little importance, if the point chosen is in the proximal head or corresponds to the normal point of entrance of the nerve, or is in the belly of the muscle. Function returns less completely, or at least less quickly, if the implantation is made near the tendinous expansion.

16. Serra, Giovanni: Chir. d. org. di movimento **8**:301 (March) 1924

Causalgia Treated by Modified Periarterial Sympathectomy.—Wood [17] reports a case of causalgia of the right hand following a crush of the middle finger. The amputation of this finger afforded only temporary relief, the pain returning in the hand and index finger. The brachial artery was exposed under local anesthesia. Manipulation of the artery increased the pain in the hand. The coat of the artery was then injected with a ring of rectified spirits (alcohol), and a white wheal formed completely encircling the artery. The next morning, the pain, tenderness, and hyperesthesia had entirely disappeared. No definite statement as to the permanence of this relief is made.

Periarterial Sympathectomy.—Mitchell [18] reports nine cases in which periarterial sympathectomy was performed on seven patients, twice on the brachial artery and seven times on the femoral artery. All of these patients had the general type of Raynaud's disease and two years had passed since the operation in one case. After the operation done on both femoral arteries, pain practically ceased, and sluggish ulcers began to assume a healthy appearance. The patient, who was practically bedridden, was able to get about in comfort, and he has remained much improved throughout the two years since the operation. The other cases are of too short duration since operation to justify any conclusions as to the permanency of the results, but they have all been relieved of pain. Mitchell believes that he is justified in recommending this operation as offering a means of relief in many trophic and vasomotor disturbances of the extremities for which in the past we have been able to do so little.

[Ed. Note.—There are many conflicting reports as to the value of periarterial sympathectomy, or at least as to the permanence of the relief afforded by this procedure introduced by Leriche. It is evidently unfair to expect it to cure certain circulatory disturbances in which there are pathologic changes within the blood vessels, as in Buerger's disease. Undoubtedly, the procedure has been employed in many unsuitable cases. In Reynaud's disease, on the other hand, great improvement, amounting to at least temporary cures, has followed periarterial sympathectomy. If our anatomic teaching is correct, it is hard to understand how the removal of the outer coat of a vessel, which deprives this small area of its sympathetic nerve supply, can result in the permanent dilatation of the whole vessel or cause it to supply indefinitely the part distal to the operation with more blood. If the sympathetic filaments passed to the vessel wall all in one region and ran distally along the vessel and its branches, the operation might be expected to deprive the

17. Wood, W. Q.: Edinburgh M. J. **31**:329 (May) 1924.
18. Mitchell, J. F.: South. M. J. **17**:609 (Aug.) 1924.

distal portion of a vasoconstrictor mechanism, but we are taught that the sympathetic filaments pass to the vessels at many different levels throughout their course; and therefore it is hard to understand why depriving a small portion of the vessel of its sympathetic nerve supply should result in a permanent dilatation of the whole distal portion of the vessel, though it might conceivably interfere sufficiently with the vasoconstrictor mechanism to bring about a temporarily increased blood supply. We have seen no reports nor have we observed any aneurysmal dilatations of the vessels thus deprived of their outer coats, but such an eventual possibility may be worth consideration.]

SYPHILIS

Bone Lesions.—Scott [19] has been impressed by the fact that, as in other tissues, the reaction of bone to the spirochaeta pallida varies. It depends on the virulence of the organism and the relative immunity of the patient. As age advances, other factors enter in, such as secondary infection, direct trauma or surgical intervention. Although certain types of congenital syphilitic bone lesions present a more or less consistent and characteristic picture, they are by no means invariable. This common disease often manifests itself in bizarre forms.

Menard and Mozer,[20] after a long and careful clinical study of syphilis, have concluded that the most frequent bone lesion of syphilis is hyperostosis. These hyperostoses have a general distribution and a predilection for long bones. The cortex becomes much thickened and encroaches on the medullary space. Hyperostosis, localized on the inner third of the clavicle, is almost pathognomonic, as is osteitis of the cranial and nasal bones. These authors have found no characteristic signs by which syphilitic spina ventosa may be distinguished from the similar lesion of a tuberculous infection.

Leri and Cottenot [21] have encountered, in one month, four cases of syphilitic osteitis of the skull during routine roentgenographic examination for various nerve lesions. This osteitis cleared up quickly under antisyphilitic treatment, and the authors suggest that these lesions will be more commonly discovered if they are carefully sought.

Joint Manifestations of Syphilis.—Fatou,[22] whose experience with syphilitic lesions is large, has noted that, in hereditary syphilis, the dis-

19. Scott, C. R.: Bone Lesions of Congenital Syphilis, Am. J. Dis. Child. **28**:38 (July) 1924.

20. Menard, V., and Mozer, M.: J. de. méd. de Paris **42**:523; 587, 1923.

21. Leri, A., and Cottenot: Bull. et mém. Soc. méd. des. hôp. de Paris, April 3, 1924, p. 432.

22. Fatou: Bull. méd., June 28, 1924, p, 735.

ease may be latent for from fifteen to twenty years and then flare up in a virulent infection. The chief characteristics of these late fulminating cases are (1) purulent fluid in the joints; (2) usually symmetrical lesions; (3) the predilection for the larger joints; (4) the merging of the acute phase into the chronic phase, and (5) a positive Wassermann reaction in the joint fluid even when negative in the blood.

Flandin [23] reports four cases of articular bony lesions of common aspect belonging in the class of chronic arthritis without any special characteristic lesions to suggest that they were of syphilitic origin. Their true nature was discovered by the concomitant presence of other lesions of a definitely syphilitic nature, and by the fact that they disappeared under antisyphilitic treatment. Flandin concludes that:

1. Old syphilis of slight virulence may produce osteartícular lesions without specific characteristics which might distinguish them from lesions caused by chronic infection or intoxication.

2. Although nonspecific in appearance, these lesions due to inherited or latent syphilis require specific therapy for improvement.

Pagniez and Coste [24] report a case of acute secondary infection of a Charcot ankle in which the organism proved on aspiration of the joint to be the bacillus of typhoid. Although the condition was acute and had been present for a month and the aspiration showed actual pus to be present in the joint, the symptoms subsided after treatment by typhoid vaccines. The authors have reviewed the literature and find that open drainage is rarely necessary in the acute flare-up of a Charcot joint, and that the fluid obtained from such joints is usually sterile.

OSTEOMYELITIS

A Clinical Lecture on Osteomyelitis.—Ashhurst,[25] in a very instructive clinical lecture on osteomyelitis, presents several important points as to its treatment. He believes in the earliest possible operation for this condition, and considers it necessary not only to incise the periosteum, but also, in every case, to open the medullary cavity and to open it sufficiently to reach the extent of the infection. He believes that guttering operations are not necessary if the cases are seen and treated properly early enough. In the late stages, the proper operation should be done after from eight to twelve weeks. He believes that, at this time, curettage should not be done. The proper operation is to employ an

23. Flandin, G.: Bull. et mém. Soc. méd. d. hôp. de Paris, Dec. 14, 1923, p. 1732.

24. Pagniez, P., and Coste: Bull. et mém. Soc. méd. d. hôp. de Paris, Nov. 9, 1923, p. 1473.

25. Ashhurst, A. P. C.: Internat. Clin. **2**:248, 1924.

Esmarch tourniquet and to remove all sequestrums, carefully saving the involucrum. The edges of the cavity must be cut off so that the bone is saucerized. He believes strongly in the benefit of iodoform bone wax, and recommends the swabbing of the cavity with 5 to 10 per cent. formaldehyd before the Mosetig-Moorhof wax is applied to the bone.

ARTHRITIS

Sulphur Treatment of Chronic Arthritis.—Meyer-Bisch [26] makes a further report on the treatment of chronic arthritis with sulphur. He explains the effect of the drug by its action on the metabolism. The blood changes consist in alterations in the white corpuscles, like those following protein therapy, and in the differences in the albumin sodium chlorid and sulphuric acid content. The urobilinuria and glycuronuria observed in the urine for a few hours after the injection of sulphur indicate its influence on the liver function. These effects are demonstrable with very small doses of three or four milligrams. He recommends the colloidal preparation of Heyden. He believes that he has seen remarkable results in chronic arthritis, but warns against too large doses which aggravate the symptoms and bring about complications.

Monoghan and Garai,[27] believing that the varying immunity to pyogenic organisms and predisposition to arthritis may be caused by disturbances in the metabolism of sulphur and the consequent disturbance of the nitrogen body chemistry, have employed, intravenously, a preparation of activated colloidal sulphur, known as 338, possessing an electric charge which renders it highly active chemically and physiologically. The writers have given over 600 injections with no accident or serious reaction. Five cases of arthritis deformans, acute polyarthritis, subacute polyarthritis, chronic polyarthritis, all with some bacteremic infection, have been favorably affected.

Reimann and Pucher,[28] on the other hand, have studied a series of cases subjected to the sulphur treatment, advocated by Meyer-Bisch, in 1921 and 1924. They find that neither the chemical data nor his clinical observations can be entirely confirmed. Favorable clinical results were observed only in cases of arthritis without gross bony changes obstructing motion. They conclude that the use of sulphur preparations in the treatment of arthritis gives results very similar to those obtained by the parenteral injections of nonspecific proteins and that, on the present evidence, it is doubtful whether sulphur has any specific curative action in arthritis deformans.

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26. Meyer-Bisch, R.: Erkrankungen des Bewegungsapparates **1**:9, 1924.
27. Monoghan, W. J., and Garai, F.: M. J. & Rec., July 16, 1924, p. 75.
28. Reimann, H. A., and Pucher, G. W.: Am. J. M. Sc. **168**:77 (July) 1924.

Focal Infection and Localization.—Nakamura,[29] working in the Mayo Clinic. has studied the bacteriology of extirpated tonsils and the localizing power of bacteria isolated in a series of selected cases of arthritis and ulcer of the stomach. The tonsils of patients suffering from ulcer of the stomach and arthritis commonly harbor streptococci, which tend to localize in the mucous membrane of the stomach and in the joints of animals, producing ulcers and arthritis. This is not true of streptococci found in the tonsils of normal persons. It may be concluded, therefore, that foci of infection harboring streptococci possessing localizing power may be important factors in the primary cause and persistence of ulcer of the stomach and arthritis.

Swaim,[30] after a very wide clinical experience in the treatment of chronic polyarthritis, is convinced that however much foci of infection may be responsible for the early symptoms, the problem soon becomes one of physiology and biologic chemistry. The judicious removal of demonstrable foci may be justifiable, but the author has observed much harm result through untimely and ill advised removal of questionable foci. In the so-called atrophic form, the acidity of the secretions seems to be increased, and the value of frequent sweat baths of low temperature, after the method of Wilde, continued over periods of months, seems to be unquestioned; they stimulate the dry skin of these patients to proper function. He believes that improvement, in these cases, is due more to a betterment in general body function than to any special diet, and urges the important corrective exercises, positions of rest and general attention to body mechanics, in raising the resistance of the patient and bringing about a more normal physiologic chemistry. The author places great confidence in Pemberton's work, but believes, as indeed does Pemberton, that the dietary treatment must, in most cases, be supplemented by other measures to bring the disease under control.

Arthritis in Asthma and Other Forms of Allergy.—Lintz,[31] after the observation of some 200 cases, has been impressed with the frequency of vague pains in the extremities of asthmatic patients, which he believes are due to arthritis. Crepitation is the most conspicuous finding, and the knee joints are most commonly affected; 88.5 per cent. of his cases gave a definite history or objective findings of this type of mild arthritis.

Type II, Degenerative, Hypertrophic Ostearthritis.—Ely[32] still maintains that the second great type of chronic arthritis "occurs almost

29. Nakamura, T.: Ann. Surg. **79**:29 (Jan.) 1924.
30. Swaim, L. T.: J. Bone & Joint Surg. **6**:510 (July) 1924.
31. Lintz, W.: New York State J. Med. **24**:249 (Feb. 29) 1924.
32. Ely, L. W.: California & West. M. J. **22**:260 (June) 1924.

exclusively in persons whose alveolar processes have abscesses at the roots of their dead teeth. Young people injure their joints as much as older people, but immune from these root abscesses, they are also immune to the disease under discussion." In the absence of fracture, bone can be attacked by disease only of its contained marrow. The author is convinced that this second great type of arthritis is definitely caused by alveolar abscesses. The joint is affected through the marrow, and the cartilaginous changes are thus explained. The stiffness, pain and restriction of motion so often occurring after joint fractures, Ely believes are due to the setting free in the joint of infectious material previously locked up in the bone.

[ED. NOTE.—We are not ready to accept entirely this hypothesis of Ely. The frequent association of apical abscesses with this type of degenerative, hypertrophic arthritis is undoubted. However, we find apical abscesses in many comparatively young adults who show no signs of this form of arthritis. We also have observed patients showing these well marked typical joint changes appearing years after complete removal of the teeth in which no residual abscesses could be demonstrated. We are not aware of any evidence which has been presented to prove that the marrow of these bones shows any constant signs of infection other than the unconfirmed presence of an ameba in the cavities of the cancellous tissue close to the eburnated articular surfaces of these joints. Ely's work is most stimulating, but until more confirmation of his cause and effect theory is obtainable, we question the wisdom of the wholesale extraction of teeth, which is still too frequently, and we believe often futilely, recommended in these cases of degenerative hypertrophic arthritis.]

Surgical Treatment of Ostearthritis.—Page,[33] writing on the surgical treatment of ostearthritis, fails, we believe, in clearly indicating the types of surgical interference appropriate to the types of chronic arthritis which he includes under ostearthritis. He says that the primary indications for recourse to radical operation are persistent pain and progressive deformity, but that the operation should not be undertaken if there is evidence of existing active infection. Four radical procedures are considered: (1) excision; (2) erasion; (3) arthroplasty, and (4) synovectomy.

Arthroplasty, the author very properly considers of almost no value in cases of ostearthritis, meaning, we take it, in all forms of chronic polyarthritis. The aim of his excisions is to remove diseased joint surfaces, and the result which he expects in the hip, elbow and wrist is a

33. Page, C. M.: Brit. J. Surg. **12**:152 (July) 1924.

pseudarthrosis; in the knee an arthrodesis. In the hip, in young or middle-aged patients, he considers a complete excision of the joint to produce a firm ankylosis is indicated; in elderly patients, a simple excision of the head. He forestalls the subsequent adduction deformity in these cases by avoiding damage to the gluteal muscles, and by tenotomizing or partially sectioning the adductor tendons. His erasion consists of removing the remaining articular cartilage and underlying and exposed sclerosed bone, and from this procedure he expects a bony or fibrous ankylosis. Some of the author's cases he reports had quite good (?) range of motion. Synovectomy, he considers an essentially conservative operation, only successful when little or no change has taken place in the articular cartilage, and therefore only successful in the early stage of the disease. He says that the aborescent lipomas and hypertrophied villi in the knee joint give the most satisfactory results.

[ED. NOTE.—We fear that this article may be misleading, as we have suggested in the foregoing. The author is evidently not limiting his surgical intervention to either Type I or Type II, for he speaks of sclerosed bone beneath the degenerative changes in the cartilage, and of the ostearthritic changes in the hips of elderly subjects. These are evidently cases of Type II, degenerative, hypertrophic, nonankylosing ostearthritis (as we use the term). He also speaks of the infectious form of arthritis in young adults and of extensive disabling proliferative synovial changes which occur only in Type I—proliferative, atrophic, ankylosing polyarthritis, in which focal infection has been so much emphasized. Now, the surgery appropriate to these two types seems to us to be very different. In Type I, if the infective process is overcome and the articular cartilage is intact, as the author states, synovectomy and the removal of the hypertrophied villi may be most effective, and later, after the cartilage has been affected, excisions or erasions may bring about a firm and satisfactory bony ankylosis, which is the ordinary terminal result of the unarrested disease. In Type II, however, as Nichols and Richardson have shown, the primary changes are not in the synovial membrane, but are in the articular cartilage, which, after its fibrillation and destruction, leaves a sclerosed and eburnated joint surface and a poorly nourished cancellous bone beneath, with cavity formation in it. In joints of this type in elderly people, with unimportant synovial membrane proliferation, synovectomy has no place, and it is difficult, from the nature of the bone, to bring about by surgery or any other means a firm bony ankylosis, after even the most thorough excision. It is in these cases, as the author suggests, that simple excision of the head of the femur is sometimes indicated to relieve pain and restore motion,

even at the expense of stability. Unless we appreciate the essential pathologic changes in these two types, our surgery will be likely to be ill conceived and our results disappointing.]

Calcium Metabolism in Arthritis Deformans.—Mark [34] finds the normal value of calcium in the whole blood to be between 10 and 12 per cent. In three cases of "arthritis deformans" (supposedly Type II), a hypercalcium percentage of 18 to 27 was found. Rigid limitation of sodium chlorid intake reduced the chlorid content of the blood and urine, but the analysis of the calcium content showed no change, nor was there any change in the clinical condition. A calcium poor diet reduced the percentage of blood calcium, but not to normal. Administration of acids, alkalis, and salicylates changed the ratio of calcium excretion in the urine and the feces, but did not alter the balance on the calcium poor diet.

NEOPLASMS

Giant Cell Tumor of Bone.—Bloodgood [35] has analyzed carefully all his material, which means an investigation of 177 cases of giant cell tumor of the bone. His study has resulted in several interesting observations. For instance, he believes that if a surgeon explores a central bone tumor in which roentgen-ray examination, palpation and exploration exclude tumor tissue outside the bone shell, he may be certain that he is dealing with a tumor resembling, in the gross material and in microscopic section, osteogenic sarcoma of the chondromyxomatous type. This tumor is easily distinguished from giant cell sarcoma and from ostitis fibrocystica. Such a statement as this coming from Bloodgood carries considerable weight and simplifies diagnosis, because the cellular pathology of giant cell tumor has heretofore been confusing to experienced pathologists, and this confusion, resulting in incorrect diagnosis, has led to many amputations and mutilating resections. Mutilation is especially great when a resection is done on the jaw. Bloodgood states that he was unable to find, in this series of cases, a central bone lesion which had an intact bone shell, and which resembled giant cell tumor in the gross or microscopically, which has not remained free from metastases. All the cases in his series were free from metastases after curetting of the tumor.

Giant Cell Sarcoma of the Femur (Epulis Type) with Metastasis in Femoral Vein.—Turner and Waugh [36] have described a case of giant cell sarcoma of the femur, of the epulis type, which occurred in the

34. Mark, R.: J. Metabolic Res., July-August, 1923, p. 135.
35. Bloodgood, J. C.: Surg., Gynec. & Obst. **38**:784 (June) 1924.
36. Turner, W. G., and Waugh, T. R.: Ann. Surg. **78**:846 (Dec.) 1923.

distal extremity of the femur, and which metastasized to the wall of the femoral vein. They believe that this tumor observed by them is of a type which is generally held to be local and benign, but that it may metastasize and become malignant. The diagnosis of giant cell tumor was made by roentgen-ray examination, microscopic examination, and, at necropsy, after death which occurred following pulmonary embolism.

Roentgen-Ray Treatment of Bone Sarcoma.—Palmer [37] has written on the roentgen-ray treatment of bone sarcoma, and believes that the roentgen ray should be applied at the earliest possible moment, regardless of the pathologic condition, it only being necessary to exclude osteomyelitis from diagnostic consideration. He believes that the dosage of roentgen rays should be left to those who are fitted to prescribe a proper dose in a case of bone sarcoma. He reports two cases, one of malignant or central sarcoma, in which roentgen-ray treatment showed the possibility of bone regeneration and definite improvement, until metastases occurred in the lung. A second case was one of giant cell tumor, and this showed, on roentgen-ray examination, the formation of new bone.

Lipoma of Thigh.—Speed [38] reports four cases of lipoma of the thigh. Clinically, lipoma or lipomyxoma of the thigh is of slow growth. Its surface is smooth, and is rarely lobulated. It is movable, and is rarely tied down by overlying adjacent tissues, which it may displace and infiltrate. Such tumors give, to the examining hand, a feeling of fluctuation; there is neither pain nor tenderness. Glancing at such a tumor may lead to a hasty opinion as to its malignant characteristics. Over the tumor are usually seen dilated veins. Such tumors may be clinically classed with the sarcomas, but, in reality, they are lipomas and probably benign.

Multiple Myeloma of Children.—Berkheiser [39] reports two cases of multiple myeloma, one in a boy, aged 12½ years, who died, and one in a girl, aged 3½ years, from whom a specimen was taken. In the case of the boy, it was possible to do a careful necropsy. Berkheiser believes that multiple myeloma may occur in children as well as in adults. He believes that backache and bone pains of obscure origin suggest multiple myeloma. Absence of Bence-Jones protein does not eliminate myeloma from diagnosis, nor is the presence of Bence-Jones protein pathognomonic. Characteristic protein is found with great frequency in patients of greater age. The lesions of children are larger than those in adults, while, in the latter, the lesions are more generally distributed.

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37. Palmer, M. B.: Am. J. Roentgenol. **11**:550 (June) 1924.
38. Speed, Kellogg: Lipoma of Thigh, Arch. Surg. **8**:819 (May) 1924.
39. Berkheiser, E. J.: Multiple Myelomas of Children, Arch. Surg. **8**:853 (May) 1924.

Lesions in the skull may perforate the outer table, or the inner table of the skull may be eroded, resulting in greater cranial pressure simulating brain tumor. If the portion of the skull over the motor cortex is involved, there may be mechanical irritation of the motor area, with clinical symptoms simulating tuberculous meningitis. Roentgenograms should be taken in cases in which tuberculous meningitis is clinically diagnosed, especially when the organisms are not found in the cerebrospinal fluid. Bilateral exophthalmos may be present in cases in which the skull is involved. Patients with multiple bone tumors, especially with vetebral lesions, can be made most comfortable by placing them on posterior molded plaster shells.

SCIATICA AND LOW BACK PAIN

Sciatica.—Jaquerod [40] states that he has been able to cure his own sciatica, as well as that in others, on several occasions by a simple manipulation. He discovered it by accident while riding horseback, and then studied the movements in order to know how to repeat them. The maneuver is carried out while reclining on a hard surface, bracing the foot against the foot of the bed. The entire leg is rotated inward in extension, while the trunk is, at the same time, rotated in the opposite direction, so that the patient looks back over the shoulder on the affected side. He advises its trial, saying that it can do no harm.

Incidence and Significance of Sacralized Last Lumbar Transverse Process.—Moore,[41] studying the incidence of sacralization of the last lumbar transverse processes or lumbarization of the first sacral segment, which cannot be distinguished, finds that these anomalies exist in 6 per cent. of all persons and in 61 per cent. of persons with lumbar symptoms. He thinks that resection promises much in selected cases, and that the person with a sacralization has a liability to back strain and should follow an occupation free from such strains.

RICKETS AND DEVELOPMENTAL BONE DISEASES; OSTEITIS DEFORMANS

Etiology of Rickets.—Park [42] has reviewed the literature on rickets, from 1650, when Glisson first established the disease as a clinical entity. His concluding sentence is worthy of quotation: "Rickets is indeed a price paid by man for his abandonment of a life in the open air for a life in houses and a diet of denatured foodstuffs: it is a sign of the operation of the immutable law of nature that nothing out of accord with her shall flourish."

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40. Jaquerod, M.: Rev. méd. de la Suisse. Rom. **44**:253 (April) 1924.
41. Moore, Sherwood: Radiology **2**:237 (May) 1924.
42. Park, E. A.: Physiological Rev. **3**:106 (Jan.) 1923.

Mellanby,[43] in an interesting article, discusses the deficiency diseases, especially rickets. He points out that the most potent dietary factor in stimulating the formation of bone is vitamin A or a subsance having a similar distribution and properties. Generally speaking, the more food a growing animal eats, the more rapid is its growth. There is always a race between the growth of free bone tissue and its calcification. Certain foodstuffs actually seem to prevent calcification independently of their effect on growth. Cereals belong to this class, and of the cereals oatmeal appears to have the greatest anticalcification effect, while white flour and rice have the least. Oatmeal contains the largest content of calcium and phosphorus, and yet, as a food, opposes most strongly the retention of these elements in the body in the form of calcium phosphate in bone. This cereal effect can be completely negatived by the antirachitic vitamin contained in cod liver oil, milk, egg yolk, and butter. Sunlight and ultraviolet radiations increase the effectiveness of the fat soluble vitamin stored in the body. Mellanby says that a proper diet can more than counterbalance darkness or absence of ultraviolet rays, but sunlight or other radiations cannot make up for a really defective diet.

Late Rickets and Osteomalacia in India.—Hutchinson and Stapleton [44] have made interesting observations on forty cases of late rickets and forty-nine cases of osteomalacia occurring in high and low caste Hindu and Mohammedan subjects in India. Histologic, roentgenologic and metabolic studies were made. They conclude that:

1. In osteomalacia, the existence of pregnancy does not produce symptoms differing in any essential way from those occurring in non-puerperal cases. Pregnancy is of subsidiary importance in its causation, and its existence does not ipso facto warrant the diagnosis of osteomalacia if a bone disease is present.

2. The existing differentiation of early rickets, late rickets and osteomalacia by age incidence would appear to be unjustifiable. The evidence points to the conclusion that these conditions are identical and represent rickets at different ages.

3. The primal causative factor in all these conditions is considered to be environmental. While lack of sunlight is of importance, lack of muscular activity is probably extremely important also.

Protti,[45] having previously discussed the fact that the administration of strontium salts to animals produced changes in the bones similar

43. Mellanby, E.: Brit. M. J. **1**:895 (May 24) 1924.

44. Hutchinson, H. S., and Stapleton, G.: Brit. J. Child. Dis. **21**:18 (March) 1924.

45. Protti, G.: Chir. d. org. di movimento **8**:470 (June) 1924.

to those seen in rickets, has now studied the time of elimination of this salt. In the bones of young animals in which strontium intoxication had been brought about, he was able to find traces of the salt fifty days after its administration had ceased. His investigations led him to infer that as long as strontium salt is present, the osteoid trabeculae calcification does not take place.

Osteochondritis of the Pubis.—Van Neck [46] reports two cases of osteochondritis of the symphysis pubis occurring in girls, aged 8 and 11, on whom he operated. No trace of tuberculosis was found, and there has been no recurrence since. He considers the affection to be similar to Köhler's disease, Legg's disease and analogous disturbances in rapidly growing bones.

Osteochondritis Deformans Juvenilis.—Galvin [47] contributes an excellent review of the literature of this disease, and after weighing the evidence concludes that the affection is probably of traumatic origin, due to a disturbance of the arterial supply about the epiphysis, although an infection may be superimposed on the original process.

McWhorter [48] reports a case resembling this condition, which seemed to be the result of an infectious process. The clinical symptoms were characteristic of an acute osteomyelitis of the neck of the femur, and an operative culture of this area produced a growth of *Staphylococcus aureus*. There was a positive focus of infection in the maxillary sinuses from which the same organism was isolated.

Essential Coxa Vara and Osteochondritis.—Guillemin [49] agrees with Froelich that the processes in essential coxa vara and osteochondritis are identical, the former affecting the epiphysis and neck of the femur, which, yielding, causes displacement of the head, while, in the latter, the process is confined to the head, and weight-bearing results in fragmentation. The inflammatory theory of etiology is favored.

[Ed. Note.—Legg and the other defendants of the traumatic theory of etiology are probably willing to admit that an acute or even subacute epiphysitis may produce changes in the neck and osseous center of the head resembling, roentgenologically, the changes seen in the disease originally described by Legg and Calvé. We have personally observed such changes. This does not prove, however, that the peculiar and very characteristic symptom complex which they have described

46. Van Neck, M.: Arch. france-belges d. chir. **27**:238 (March) 1924.
47. Galvin, A. H.: Surg., Gynec. & Obst. **38**:58 (Jan.) 1924; ibid. **39**:2146, 1924.
48. McWhorter, C. L.: Surg., Gynec. & Obst. **38**:632 (May) 1924.
49. Guillemin, A.: Rev. d'orthop., January, 1924, p. 51.

is due to an infection. The whole history and almost spontaneous resolution of the disease argues against an infection. In at least one very typical case, not only were cultures entirely sterile, but histologic examination of a small block of bony tissue, including portions of the head, epiphysis and neck, revealed no microscopic evidence of an inflammatory process. On exposure, the shape and surface of the articulation of the head appeared normal, in spite of the changes in the osseous center shown in the roentgenogram.]

Osgood-Schlatter Disease.—Van Neck,[50] after a study of six cases of this condition, advances the theory that it is due to a tenosynovitis of the patellar tendon similar to the common tenosynovitis of the Achilles tendon. He has found, at times, tenderness throughout the course of the tendon and a calcereous deposit near the attachment of the tendon to the patella, as well as at the tibial tubercle.

Koehler's Disease of the Second Metatarsal Head.—Meyer[51] has collected from the literature forty-eight cases of the so-called Koehler's disease, and adds two of his own. The average age was about 27 years, and it occurred in females about three times as often as in males. He recognizes three stages: (1) subchondral changes of structure; (2) widening and flattening of the head and widening of the articular space, and (3) osteophytic changes and perhaps free body formation, with changes of the base of the proximal phalanx. He inclines to the etiologic theory of repeated slight trauma, and believes that the treatment should, in most cases, be conservative. He thinks the condition is analogous to Legg's disease of the hip and the lesions of the developing tibial tubercle.

[ED. NOTE.—We believe that repeated slight traumas by external violence or strong muscle pulls on developing centers of ossification offer probably sufficient explanation of these variations from the normal in the femoral head, the tibial tubercle, the os calcis, the second metatarsal head, the lower end of the radius and the olecranon, which Allison has pointed out resemble each other closely, and which are confusingly called the disease of the man who first (though sometimes not the first) described them. At all events, they become entirely quiescent and often go on to complete cure without operative intervention, and do not behave like infections of the bone or tendons. In the lesions of the adolescent tibial tubercle, there is generally only sharply localized tenderness over the tubercle, and, before excess of calcium deposit is seen, a wider space is usually evident in the roentgenogram of the

50. Van Neck, M.: Arch. franco-belges de Chir. **26**:1060 (Nov.) 1923.
51. Meyer, Moritz: Beitr. z. klin. Chir. **130**:655, 1924.

affected knee, between the shaft of the tibia and the tongue-like process of the developing upper epiphysis of the tibia, than on the normal side.]

Mono-Osteitic Type of Paget's Disease; Osteitis Deformans.—Newton [52] reports three cases of this condition. The author summarizes by saying that "Paget's disease of bone (osteitis deformans), an unmistakable disorder in its outspoken form, is, nevertheless, a rare disease. The so-called mono-osteitic type is still more uncommon. When, however, it appears, it presents in a single bone exactly the same clinical picture and roentgenologic findings which are present in the generalized form." Until more information is forthcoming, we should probably consider these cases only as a localized expression of the better known generalized disease.

BONE AND JOINT SURGERY

Spasmodic Torticollis.—McKenzie [53] reports an operation for relief of spasmodic torticollis which was performed by Cushing. It consisted of a combined cerebellar and upper cord exposure, with intrameningeal division of the roots of the spinal accessory nerve and of the anterior and posterior roots of the upper three cervical nerves. The result, one year after operation, was complete paralysis and atrophy of the sternomastoid, weakness and atrophy of the upper trapezius, with slight drooping of the shoulder. The splenius, however, stood out with some prominence and appeared to be in mild spasm, and the posterior muscles, on the opposite side of the neck, also gave the impression of slightly increased tonus. The deeper muscles on the right, supplied by the first, second and third cervical nerves, were probably paralyzed, although this could not be ascertained definitely, leaving the partly paralyzed splenius under the control of the lower cervical nerves. The writer states that the result proved successful beyond expectation. He interjects a word of caution, however, saying that this may turn out to be an arrest, rather than a cure.

[ED. NOTE.—Operations for the relief of spasmodic torticollis have come and gone, and we are still reaching for the means of alleviating this intractable condition. Finney has succeeded in obtaining some real benefit by the extraspinal division of the roots of the upper cervical nerves. The intrathecal route, as devised and practised by Cushing, would seem an improvement on the method of Finney, and, at least in the hands of its inventor, a simpler and better method of approach. If

52. Newton, F. C.: Mono-osteitic Type of Paget's Disease of Bone (Osteitis Deformans), Arch. Surg. **8**:24 (Jan.) 1924.

53. McKenzie, K. G.: Surg., Gynec. & Obst. **39**:6 (July) 1924.

the spasm fails to stop after the division of the main motor supply in this manner, it will be evidence that we must look elsewhere for its point of origin.]

Treatment of Muscle and Joint Injuries by Electricity.—Smart [54] describes a special electric coil by means of which it is possible to cause graduated contractions of any muscle or groups of muscles. The contraction, so produced, is claimed to be a wave contraction of the entire length of the muscle, rather than a local spasm of part of the muscle, as is produced with the ordinary faradic coil. The author advises its use in muscle and joint injuries within the first twenty-four hours, and hopes by this means to improve the blood supply and prevent stagnation He considers the results as far superior to those obtained by massage and passive motion.

[ED. NOTE.—Smart was, we believe, one of the originators of the so-called "Bristow" coil, a device which, both for diagnostic and therapeutic purposes, has stood the test of time.]

Arborescent Lipomas of Tendon Sheaths.—White [55] reports two cases of the rare condition arborescent lipomas of the tendon sheaths. Clinically, these cases showed diffuse soft elastic swellings, without clearly defined edges, but with a fine lobulation in the course of some of the tendons. One case was of from seven to eight years' duration, and the condition was associated with bad deformities of the feet and toes. In this case, some of the villi proved as large as marbles and as dense as cartilage. Both cases were relieved by operation, and there was no recurrence of the condition.

Penetrating Wounds of the Dorsal Surfaces of the Fingers.—Polloson and de Rougemont [56] call attention to the necessity for careful scrutiny of wounds of the dorsal surfaces of the fingers. When a blow is received or delivered with the hand clenched, the integuments over the joints of the fingers are tightly stretched and offer but slight resistance. A penetrating wound of the joint may easily be produced, or the extensor tendons may be ruptured. In studying a series of cases, they have been struck by the number of infected joint wounds or of ruptured tendons caused in this manner.

Carpal Ganglion.—Neumüller and Orator [57] have been trying out a new method of surgical treatment for ganglions of the carpal region. After exposure, they opened the ganglion by a crucial incision, turned

54. Smart, M.: Brit. J. Radiol. **20**:77 (March) 1924.
55. White, J. R.: Surg., Gynec. & Obst. **38**:489 (April) 1924.
56. Polloson, E., and de Rougemont: Presse méd., March 26, 1924, p. 515.
57. Neumüller, H., and Orator, V.: Deutsche Ztschr. f. Chir. **186**:69, 1924.

back the four flaps and fastened them in the subcutaneous space. They thus established a connection between the cyst cavity and the subcutaneous lympathics, and recurrence was prevented. They feel that a better method is needed, as Kuettner's figures showed recurrence in 50 per cent. after puncture and squeezing, and in 30 per cent. after complete excision. They believe that the tendency to recurrence is fully explained by pathologic studies, which show that a ganglion is not merely a widening of the articular tube, but a cystic degeneration in the wall of the capsule.

Thrombosis of the Axillary Artery.—Auvray [58] reports a case of thrombosis of the axillary artery complicating a case of fracture of the upper end of the humerus, six days after injury. There was ischemia of the hands and fingers, a swelling at the juncture of the axillary and brachial arteries, and palpable pulsation at this point. The artery was opened, the thrombus removed, and the artery sutured laterally. The gangrene ceased; the circulation returned to the fingers, and amputation was not necessary.

Arm-Chest Adhesions.—Davis,[59] in an article on arm-chest adhesions, calls attention to the necessity of reconstituting the axillary space. He considers this the key to the problem. One operation is seldom sufficient to obtain a cure, and usually it is a matter of an elaborate plastic procedure with several stages. He reports good results in a considerable series of cases.

Operative Fixation of the Spine.—Lange [60] reports the technic for operative fixation of the spine, which he has employed since 1920. He implants two celluloid rods, from 5 to 10 mm. in thickness, one on either side of the spinous processes. These are tightly fitted to the spines by means of thick silk threads passed through drill holes. Since 1920, he has operated in fifty-two cases. The results are considered excellent. In five cases, it was necessary to remove the rods, but Lange thinks that with better technic this complication may be avoided. He is convinced that celluloid causes but slight irritation of the tissues. The leukocytic reaction is small and only a thin covering of connective tissue is formed.

[ED. NOTE.—We must confess to a certain feeling of amazement on reading Lange's report of his work with celluloid implants. In the present state of surgical progress, there is neither any need nor is there

58. Auvray, M.: Paris méd., March 15, 1924, p. 258.

59. Davis, J. S.: Arm-Chest Adhesions, Brachio-Thoracic Adhesions. Axillary Webs, Arch. Surg. **8**:1 (Jan.) 1924.

60. Lange, F.: Ztschr. f. orthop. Chir. **45**:492. 1924.

any possible advantage in the use of nonabsorbable splints for the internal fixation of the spine. The experience with Albee's bone graft operation and the various fusion operations, such as Hibbs' or Forbes' operations, covers a period of nearly fifteen years, and the results have been eminently satisfactory. In justification of Lange, it should be said that he was a pioneer in this field. He operated on a patient as early as 1902. His early work was with metal splints, and it is only recently that he has used celluloid. He has therefore been consistent.]

Surgical Exposure of the Humerus and Femoral Shaft.—Henry [61] has made a study of the anatomic relations of the humerus and femur in an endeavor to determine the best methods of surgical approach. His conclusions and the reasons for them are given in minutiae, and should be studied in the original article. For the exposure of the shaft of the humerus, he advises an incision on the lateral aspect of the arm, following the course of the cephalic vein from the tip of the coracoid to the bend of the elbow. The deep fascia is divided along the outer edge of the belly of the biceps and the bone reached along the intramuscular plane. Partial exposure may be obtained by shortening the incision. He also describes extensions of the incision upward, to reach the shoulder, and downward, to approach the elbow joint. The usual approach to the femur is along the lateral surface of the thigh. This incision is inconveniently placed and cuts several perforating arteries of the vastus internus. The incision described by the author is nearly bloodless. The skin and deep fascia are divided on a line from the anterior iliac spine to the outer angle of the patella. A little below the greater trochanter, the rectus is easily separated by blunt dissection from the vastus externus. In the lower third, the aponeurotic fibers of the vastus joining the rectus tension must be divided. He explains in detail how the lower end of the incision may be modified to fit various conditions which occur in the lower end of the femur.

Habitual Dislocation of the Patella.—Gallie and LeMesurier [62] have reported a new method of anchoring the patella to prevent recurrent dislocation. The patella is exposed through a simple longitudinal incision, and the adhesions freed so that the dislocation can be reduced. A 6 inch (15.2 cm.) length of about one-half the Achilles tendon is removed. One end is threaded through a transverse drill hole in the patella, and the other end passed through drill holes in the internal condyle of the femur. The fixation is strong and permanent and

61. Henry, A. K.: Brit. J. Surg. **12**:84 (July) 1924.

62. Gallie, W. E., and LeMesurier, A. B.: J. Bone & Joint Surg. **6**:575 (July) 1924.

mechanically satisfactory. In many of the less severe cases, a strip of fascia lata is sufficiently strong for the purpose.

[ED. NOTE.—Several of us have seen Gallie's patients, and some of us have employed his method. We are convinced that it offers a solution, especially for the intractable cases. In a former Report of Progress, we recorded a modification of the method devised by one of us (R.S.), the chief difference being that the fascial strip was implanted on the internal aspect of the upper end of the tibia instead of in the femoral condyle. The functional results have been good by both methods, and time alone will show which gives the more solid and functionally perfect fixation.]

Flexion Deformity of the Knee.—Parker [63] makes a plea for the fully extended position of the knee in the treatment of pathologic conditions involving that joint or in the correction of flexion deformities. He says that the straight knee will support the body weight either standing or walking, and that it gives the greatest length of limb with the least amount of limping.

[ED. NOTE.—We must take issue with Parker's sweeping conclusion that the fully extended position is always the optimum position for ankylosis of the knee joint. We feel that the straight position is perhaps best when there is much shortening, although even here we usually strive for about 10 degrees of flexion. In childhood, also, there is an advantage in the straight position, as, with even a few degrees of flexion, there is a tendency for the angulation to increase with growth. In the adult, the straight knee entails great awkwardness when sitting, and there are few persons who do not prefer from 15 to 20 degrees of flexion in order to render themselves less conspicuous. This slight angle makes almost no difference in length and practically none in walking. Many persons have stiff knees as a part of a generalized arthritis, and these must, perforce, lead an inactive life in which a comfortable sitting position is much more important than standing or walking. In a case of this type, the optimum functional position may be flexion of as much as 35 degrees.]

Operative Correction of the Flexed Ankylosed Knee.—Mommsen [64] considers that the "Quengel method" of correcting flexion deformities of the knee is superior to any other method which he has employed. A curved osteotomy of the lower end of the femur is performed, and then the leg is placed in a plaster spica with Hackenbruch's clamps bridging the area operated on. After ten days, extension is applied, and, very gradually, the knee is straightened without pain and without danger

63. Parker, C. A.: Journal-Lancet **44**:35 (Jan. 15) 1924.
64. Mommsen: Ztschr. f. orthop. Chir. **43**:542, 1924.

to the vessels or nerves. In a case that is cited, there was ankylosis with the knee in a position of 120 degrees of flexion, and yet complete extension was obtained within three weeks. It is important to follow a careful technic in applying, and especially in padding, the plaster casts.

Peroneal Paralysis Following Osteotomy of the Tibia.—Brandes [65] calls attention to the danger of paralysis of the common peroneal nerve following curved osteotomy of the upper end of the tibia for the correction of genu valgum. He explains the occurrence of this complication on anatomic grounds. The peroneal nerve is firmly bound down to the head of the fibula, and in correcting the genu valgum at this level the nerve may be forcibly stretched and pressed against the bone. Brandes recommends that when this operation is to be employed, the nerve be first isolated and displaced posteriorly, so that it cannot be stretched over the head of the fibula.

Dangers from Radical Operations for Bow Legs.—Martin [66] of the Berlin Surgical Clinic cites two cases illustrating the dangers that may result from some of the modern radical procedures for correcting bow legs. One patient had been operated on by Loeffler's method. This consists in the temporary resection of the deformed part of the tibia, which is then crushed into small pieces and reimplanted in the periosteal tube. Delayed union resulted, and there had been a partial recurrence of the deformity. In the second case, Kirschner's operation of multiple osteotomy of the deformed area was performed. The child died, the second day, from fat embolism. Martin advocates the more frequent use of the old linear osteotomy, which is much safer, although its cosmetic results are less perfect.

Fibula pro Tibia.—Fieschi [67] reports five cases of substitution of the fibula for the tibia when the diaphysis of the latter was absent, with excellent results in four cases. In one case, death resulted from recurrence of the old infection, although a thigh amputation was done in an attempt to arrest the sepsis. Following the substitution in the four successful cases, the leg increased in length as the children grew, overcoming in part the differences in length previously present. The transplanted bone also increased in size, so that it was capable of sustaining the weight of the body without apparatus. He urges the more permanent removal of the entire diaphysis of the tibia in cases of extensive osteomyelitis. Subsequently, the defect can be repaired by the fibular substitution. He also makes a plea for surgeons to keep this

65. Brandes: Zentralbl. f. Chir. **40**:1506 (Oct. 6) 1923.
66. Martin: Zentralbl. f. Chir. **40**:1746 (Dec. 1) 1923.
67. Fieschi, D.: Chir. d. org. di movimento **8**:213 (March) 1924.

possibility in mind in cases of extensive compound comminuted fractures of the tibia in which thigh amputation might otherwise be the treatment of choice.

[ED. NOTE.—The only fault we find with Fieschi's conclusions is that he urges resection of the tibial diaphysis for osteomyelitis. We have seen the rise and wane of this procedure, and the reasons for its abandonment are that it has given too many bad results, both as regards immediate mortality and later function.]

Tibio-Astragaloid Arthroresis.—Mezzari [68] describes Putti's operation of tibio-astragaloid arthroresis. The latter term is defined as meaning limitation of joint motion. The operation consists in the formation of a bony block at the front of the ankle joint, preventing dorsal flexion above an angle of 110 degrees. A block of bone, 3 cm. by 2 cm., is removed from the tibia and implanted in a transverse trough in the astragalus, directly anterior to the margin of the articular surface of the tibia. The block projects from the astragalus, from 1 to 1.5 cm. Its use is chiefly to stabilize a knee in which extension is weak or absent from paralysis of the quadriceps. The results are illustrated by the reports of twenty-three cases. Later, roentgenograms showed a newly formed mass of bone at the site of the implant, and the functional results were excellent, in general. In cases with lateral instability, subastragaloid arthrodesis is required, and, in many of the reported cases, other operations were performed in addition to the arthroresis.

Operation for Correction of Plantar and Adduction Contraction of the Foot.—Rugh [69] describes a procedure which he has found successful in the correction of severe types of claw foot. It is in reality a modification of the old Phelps operation. A transverse incision is made, extending from the internal malleolus downward across the sole. The fascia, the flexor brevis and the adductor hallucis are divided, and in severe cases, in addition, the tendons of the tibialis posticus and flexor longus pollicis. The astragaloscaphoid ligament may also be cut, and whatever bony deformity is present should be corrected. The wound is packed with gauze, and a plaster cast applied with the foot in overcorrection. An opening is cut over the inner and under side of the cast. From the inner side of the opposite calf, an elongated pedicle is raised, consisting of skin, fat and fascia, and this is used to close the defect. The plaster cast is extended to hold the two parts in opposition. The whole purpose of the transplant is to prevent contraction of the scar. Later, the base of the flap is divided, and this is shaped and sutured to close the defect.

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68. Mezzari, A.: Chir. d. org. di movimento **8**:431 (June) 1924.
69. Rugh, J. T.: J. Bone & Joint Surg. **6**:664 (July) 1924.

[ED. NOTE.—This seems a very severe procedure, and one fraught with many perils.]

Transplantation of Periosteal Callus.—Klinkerfuss [70] has made a very extensive experimental study, on rabbits, of the growing power of periosteal callus. The callus was obtained by first cutting into the ulna and stimulating repair and then removing the newly formed tissue, from seven to twenty-one days later, and implanting it elsewhere. He concludes from his work that callus grafts do not die, but continue growing, in contrast to solid grafts which are absorbed and replaced by new bone. Callus grafts form new bone more rapidly and in greater amount than solid bone transplants. They persist as long as solid bone grafts and become quiescent at about the same time.

FRACTURES

Fractures of the Elbow Resulting in Ankylosis.—Cohn [71] states that ankylosis of the elbow is likely to result from any of four different types of fracture. The serious nature of such injuries must be recognized at the outset and every effort made to prevent future disability, if this result is to be avoided. He groups these cases as follows:

Group 1. Epiphyseal fractures with displacement. Every effort should be made to reduce the fracture immediately, and if this fails, open operation should be employed.

Group 2. Supracondyloid fractures either comminuted or simple. Ankylosis occurs in such cases only when vicious union results. Immediate reduction prevents such a condition. When operative correction is required, Cohn employs the externolateral incision, taking pains to isolate and retract the radial nerve.

Group 3. Fractures with displacement of a free fragment, which acts like a foreign body wedged between the articular surfaces. Included in this group are fractures of the internal condyle, coronoid process and head of the radius. Cohn advises removal of the free fragment when it is impossible to replace it and maintain the position.

Group 4. Fractures of the ulna, particularly in the upper third. If reduction cannot be maintained, open operation is advisable.

Fractures of the Head of the Radius.—Driberg [72] recognizes three varieties of fracture of the radial head: (1) a fissured fracture or chipping off of the outer side of the head of the radius; (2) a crush fracture, in which the head is flattened and mushroomed, and (3) a

70. Klinkerfuss, G. H.: Surg., Gynec. & Obst. **38**:625 (May) 1924.
71. Cohn, I.: South. M. J. **17**:511 (July) 1924.
72. Driberg, J.: Practitioner **112**:262 (April) 1924.

transverse or comminuted fracture with the head completely separated and displaced. The first group should be treated by massage and movement from the first. No splint is required, only a sling. In children, the arm should be kept in full flexion and supination for a week. In the mushroom type of fracture, massage and movement should be tried for two weeks, and if the range of motion is not improving at the end of this time, operation for removal of the head should be performed. In fractures of the third variety, excision of the head is indicated as early as possible.

[ED. NOTE.—The results of excision of the head of the radius so far as restoration of the rotary movements of the forearm is concerned, are not always good. This is chiefly due to new bone formation at the site of resection. We have a definite impression that better results may be obtained if the operation is performed either soon after injury, before the osteogenetic action has begun, or late, when it has ceased.]

Neuropathy Complicating Colles' Fracture.—Jones [73] calls attention to an article by Professor Turner of the University of Leningrad on neuropathic complications of Colles' fracture. It is not uncommon to find cases of the latter condition in which the hand is swollen, especially on the dorsum, the fingers very stiff, and attempts at forced movements very painful. Turner considers this a neurotrophic condition, the result of an interference with the trophic supply of the carpus and the metacarpus. Thus when there is marked radial deviation of the hand, the cutaneous fibers of the ulnar nerve may be pressed on and irritated by the lower end of the shaft of the ulna. The dorsal interosseous nerve is more commonly involved, its terminal fibers being caught by the backward displacement of the lower radial fragment. The terminal branches of the radial nerve may be irritated in the same manner. Marked atrophy of the carpus, metacarpus and phalanges is found on roentgen-ray examination, and this is greater in amount than would be caused by simple nonuse. Treatment in these cases is required over a long period, sometimes for as much as a year. Turner states that he has been able to cure the condition and cut short the treatment by a corrective osteotomy of the radius and replacement of the broken fragment.

Colles' Fracture with Fracture of the Cuneiform.—Schinz [74] reports a case of Colles' fracture complicated by fracture of the ulnar styloid and of the cuneiform. He considers the last a fairly frequent complication, having encountered thirteen cases within a period of three years.

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73. Jones, R.: Brit. M. J., May 3, 1924, p. 798.
74. Schinz, H. R.: Zentralbl. f. Chir. **50**:1764 (Dec. 1) 1923.

He has made a careful study of the condition and states that the characteristic lesion is an avulsion fracture of a small piece of bone at the point of insertion of the dorsal radiocarpal ligament. Denecke and Bardenheuer have found, by experience, that a fracture of the cuneiform may be produced by a fall on the hand with the wrist in extreme dorsal and ulnar flexion, and with most of the force being exerted on the ulnar side of the hand. The fracture is visible in only the lateral roentgenograms, and this is the reason why it is so frequently overlooked.

Isolated Fracture of an Intervertebral Disk.—Arcangeli [75] reports a case of fracture with displacement of the intervertebral disk between the first and second lumbar vertebrae. No cord injury was present. Some limitation of the movements of the spine resulted, and the man said he was unable to do heavy work. The diagnosis was made from roentgenograms which showed normal vertebral bodies, but a narrowing of the space between the two bodies. At their anterior margins, the two bodies were almost in contact.

Isolated Fracture of a Spinous Process.—Zollinger,[76] from a personal experience with fifteen cases encountered while doing accident insurance work in Switzerland, is convinced that isolated fracture of one or more of the spinous processes in the lower cervical and upper dorsal segments is one of the common injuries of the spine. In respect of symptoms, duration and sequelae, it is not severe. It is produced by the incoordinated action of a portion of the trapezius when the person is doing heavy lifting, shoveling or like work. There is marked tenderness, often lateral deviation, crepitus and abnormal mobility. Simple therapeutic measures suffice, and complete fixation, such as would be obtained with a cast, is not required.

Fracture of the Pelvis.—Norris [77] recommends the following method of treatment for fractures of the pelvis: From the longitudinal cross bars of a Balkan frame is swung a tight sling around the pelvis. The sling is then counterpoised with sufficient weight to cause it to support the hips, but not to lift them from the bed. Whenever it is necessary to lift the pelvis, additional weights are added temporarily. The ends of the binder should be fastened to wooden bars in order to prevent wrinkling and uneven pressure on the pelvis. The advantages of the method are that it permits the muscles, which make the best kind of splint, to be

75. Arcangeli, M.: Chir. d. org. di movimento, December, 1923, p. 172.

76. Zollinger: Fortschr. a. d. Geb. d. Röntgenstrahlen. **31**:216, 1923.

77. Norris, H.: Fracture Through the Pelvic Rim, J. A. M. A. **83**:192 (July 19) 1924.

accommodated constantly to the affected part; the pressure is constant and can be easily regulated; the pain is reduced, and the convalescent period decreased.

[ED. NOTE.—Although sling suspension for fracture of the pelvis is not new, little has been written about it, and it is for this reason that we include this article in the Report of Progress. Some of us who have had personal experience with the method, are convinced that it is of real value in certain types of cases, especially those of the more severe fractures.]

Locomotion After Fracture of the Neck of the Femur.—Speed [78] reports two cases in women, aged 64 and 65, who fractured the femoral necks, but who were able to walk with only slight pain immediately thereafter. Roentgen-ray examination showed both fractures were impacted, and that the angle of the neck, instead of being decreased, was exaggerated. The treatment was rest for a few days and then protected weight bearing with a caliper splint. Speed feels that such a fracture can be produced only when the patient falls backward with all the weight thrown on the injured hip while the external rotator muscles of the thigh are violently contracted in an effort to restore the body balance.

Fracture of the Femoral Shaft in Small Children.—Poulsen,[79] for fractures of the femur in children, recommends the use of a plaster spica, which is applied while traction is being exerted on the leg and the hip is flexed to a right angle. He states that with this method he has been able to obtain good results in some cases in which overhead extension had failed to reduce the deformity.

Fracture of the Patella in Children.—Fracture of the patella in children is a very rare injury, and Hallopeau's[80] report of three cases is, therefore, of interest. The first occurred by direct violence, and the other two as a result of muscular contraction. In one, the patella was broken into four equal portions. Hallopeau employed the open method in dealing with these cases, and encircled the patella with bronze wire. He considers this technic better than other methods of suture in children.

Check Fractures.—Diemer [81] states that, in certain cases of injury without gross bone lesions, it is possible, by using a very fine focus tube with perfect exposure, to demonstrate a slight disparity of bone detail

78. Speed, K.: Ann. Surg. **79**:270 (Feb.) 1924.
79. Poulsen, H.: Ugesk. f. Læger **86**:140 (Feb. 14) 1924.
80. Hallopeau, P.: J. de méd. de Paris, Nov. 10, 1923, p. 927.
81. Diemer, F. E.: Radiology **3**:160 (Aug.) 1924.

with an indefinite but none the less distinct line of abnormality. This is considered to be evidence of a check fracture, and the findings are explained on the basis of either a slight impaction on one aspect, with extremely slight separation on the other, or an extremely slight impaction on all sides. It occurs chiefly in bones without pull bearing or weight bearing function. It is most commonly found in the lower end of the fibula, next most commonly in the skull, and next in the ribs. Check fractures are usually produced by indirect violence, a turning over of the ankle, a contrecoup, a fall on the extended arm (fracture of the head of the radius), or a blow on the chest (fracture of the ribs, posteriorly).

Ununited Fractures.—Kolodny [82] has performed a series of experiments on dogs to show the effect of disturbances of the endocrine system on the healing of fractures. Nine of the experiments were with the testicular secretion, eight with the pancreatic, and eleven with the thyroid. He concludes that the results of this work prove, beyond reasonable doubt, that endocrine disturbances play a prominent rôle in the suppression of the regenerative process in bone. He is far from insisting that these disturbances influence bone regeneration alone, but suppression of the healing of fractures is, at least, one of the results. While recognizing the part played by the endocrine system in producing nonunion in certain cases, he does not consider it the only factor, or even necessarily, a principal factor in most cases.

Campbell [83] notes that nonunion of fractures occurs more frequently now than formerly. He suggests as the explanation, first, a change in the type and nature of fractures, as evidenced by the increase in the number of cases of multiple fracture, which impose a greater demand on callus output than simple fractures; and, second, a change in methods of treatment, due to the present roentgenologist control, which necessitates more frequent manipulation than previously. He has worked out a special operative technic for the treatment of ununited fractures, which he has employed thus far satisfactorily in seventeen cases. He uses four trained assistants in order to shorten the duration of the operation. The periosteum is stripped for three fourths of an inch (1.8 cm.) on each fragment, leaving the soft parts attached. Shavings are removed from the cortex until there is a flat surface on one side from 3 to 4 inches long. A broad, flat, massive graft is taken from the tibia and split through the thin edge, leaving a strong outer plate, the cortex, and an inner one, the endosteum. The endosteal graft is placed in the medulla, bridging the seat of fracture, while the outer plate is attached

82. Kolodny, A.: Surg., Gynec. & Obst. **38**:792 (June) 1924.
83. Campbell, W. C.: Ununited Fractures, Arch. Surg. **8**:782 (May) 1924.

to the fractured bones over the flattened surface by three or four autogenous bone pegs and chromic sutures, if necessary. In some cases, the graft is fitted into a slot cut in the fractured bone. External fixation is exployed for eight weeks.

[ED. NOTE.—The more we study the problem of nonunion, the more evident it becomes that many different etiologic factors are involved. The mechanical factors, such as imperfect reduction, improper fixation and interposition of tissue, have long been recognized. The more general ones are beginning to be so. Petersen has shown the influence of altered calcium and phosphorus metabolism. Kolodny's work proves that endocrine disturbance also may play a part. Injury to the nutrient vessels, with consequent restriction in the blood supply to the injured bones, has also been put in cause. Campbell's suggestion that the organism may encounter difficulty in producing sufficient callus to meet the demands of multiple fractures is interesting, but remains to be proved. His observation on the effects of overtreatment is timely. Too much manipulation and too heavy traction may lead to nonunion, of that we are sure. The problem is physiologic as well as mechanical, and the treatment, if it is to be successful, must be approached from this basis. Before proceeding to operation, the normal physiology must be restored, and proper diet, fresh air, sunshine and active use of the injured limb are necessary preliminaries. The operation itself should be as simple as possible. In general, the osteoperiosteal graft offers the best means of transplanting cells with osteogenetic power. It may be necessary to supplement it with a massive graft when rigid internal fixation is required. The intramedullary graft should, in general, be avoided. Following operation, active use, with protection as from a caliper splint, should be begun as early as possible. The whole matter of bone repair has been in a state of confusion. We are still groping in the dark, but these ideas, we believe, have emerged into the light and may safely be used as a basis for treatment.]

Compound Fractures.—Starr,[84] writing on the treatment of compound fractures, advises a careful débridement as soon as possible after injury, but warns against the extensive removal of loose bone fragments as likely to result in nonunion. He favors gravity drainage, whenever this can be obtained, and is against any attempt at closure by primary suture. He feels that "Bipp" paste is of value when properly used, i. e., spread in a very thin layer over the entire wound. He decries the use of metal plates with screws in compound fractures. Adequate fixation may be obtained with one of the traction splints of the Thomas type.

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84. Starr, C. L.: Illinois M. J. **45**:401 (June) 1924.

Carlucci,[85] from the first surgical service of the Bellevue Hospital, reports the results of forty-two cases of compound fracture of the lower extremity, which either involved a joint or were situated in the juxta-articular region. Ten cases came to amputation: seven of the femur and three of the leg. Four of the patients who had femur amputations died. The total mortality of the forty-two cases was five. Gas infection developed in five cases, and three patients recovered. Osteomyelitis developed in seven cases. There were no cases of secondary hemorrhage. Treatment consisted of débridement, skeletal traction, overhead suspension and the usual supplementary procedures.

DISLOCATIONS

Habitual Dislocation of the Shoulder.—Hofmann[86] reports a new operative method for the relief of recurrent dislocation of the shoulder. The essential point is myotomy of the latissimus dorsi tendon at a point about 3 to 4 inches (7.6 to 10.1 cm.) from its insertion in the humerus. He has employed the method thus far in only one case, but this was in a young man who had dislocated the arm twenty-eight times within three years, often by a very insignificant motion such as removing his coat. Hofmann thinks that sudden, unguarded movement of the latissimus dorsi plays an important part in bringing about the dislocation. Tenotomy of this muscle results in inability to draw the arm backward, but this is a minor disadvantage in comparison with the constantly guarded movements which the patient is otherwise forced to make. In the rare case of recurrent posterior dislocation, tenotomy of the tendon of the pectoralis major would appear to be the logical procedure.

MacAusland[87] has made an extensive review of the literature on recurrent dislocation of the shoulder. He concludes that the majority of cases have been treated by some form of capsulorraphy, and in most of these satisfactory results have been obtained. He reports the results in five cases in which he had done a reefing of the anterior portion of the capsule, just underneath the tendon of the subscapularis. The time interval varied from nine months to eleven years, and in none had there been any recurrence.

Old Dislocation of the Os Magnum.—Thornton[88] reports an interesting operative procedure which he employed in a case of old dislocation of the os magnun. The patient was first seen six weeks after injury. The wrist and fingers were in a position of marked palmar

85. Carlucci, G. A.: Ann. Surg. **79**:779 (May) 1924.
86. Hofmann, K.: Zentralbl. f. Chir. **51**:388 (March 8) 1924.
87. MacAusland, W. R.: Surg., Gynec. & Obst. **38**:739 (June) 1924.
88. Thornton, L.: South. M. J. **17**:430 (June) 1924.

flexion and could not be extended; movements were painful, and the grasp was weak. Reduction was attempted by open operation and could be accomplished only by shaving off the cartilage from the os magnum and from the bones with which it articulated. Following reduction, bone shavings were packed into the crevices about the os magnum in order to stimulate bony union and thus stabilize the mediocarpal joint. The result was practically normal function and a strong stable wrist. Thornton suggests that stabilization ought to be the method of choice in the treatment of derangements of the carpus rather than excision of the injured or dislocated bone.

[ED. NOTE.—We believe that stabilization of the wrist as described by Thornton represents a distinct advance in the treatment of injuries of the carpal bones. The operation may be modified to meet the varying conditions of injury. Thus, in an ununited fracture of the scaphoid, the attempt should be made to support the unstable bone by fusing it to the os magnum and the latter to the semilunar. This would in no wise affect the movements of the radiocarpal joint, on which the mobility of the wrist depends.]

Traumatic Dislocation of the Hip in Childhood.—Cheyne [89] has reviewed the literature on traumatic dislocation of the hip in childhood. The condition should be considered rare, as he has been able to find only fifty-three reported cases, to which he adds six of his own. Forty-two of the cases were in males, and forty were of the dorsal, iliac type. Manipulative reduction was successful in all cases in which it was tried within fourteen days after dislocation. When open reduction was required, the most satisfactory approach was the lateral one, with subperiosteal detachment of the muscles from the great trochanter or by detachment and subsequent fixation of the trochanter. Late results, when reported, have not been wholly satisfactory. Limping and pain have often occurred. The prognosis should be guarded on account of the possibility of the later development of a pseudocoxalgia (Legg-Calvé disease).

Rotary Dislocation of the Patella.—Scott [90] reports two cases of traumatic dislocation of the patella in which the superior border of the bone was drawn into the intracondylar notch. Open reduction was necessary and, at operation, it was found that the tendon of the quadriceps was ruptured, the joint capsule extensively lacerated, and the superior edge of the patella was resting on the anterior crucial ligament. The dislocation was reduced and the damage repaired. Both patients were discharged within thirty days, with good functional results.

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89. Cheyne, C. C.: Brit. J. Surg., July, 1924, p. 52.
90. Scott, C. M.: Dislocated Patella, J. A. M. A. **82**:1198 (April 12) 1924.

AMPUTATIONS

Little [91] issues a warning against the indiscriminate amputation of paralyzed legs. He calls attention to the fact that an amputation stump with paralyzed muscles is useless, and offers much less support from the standpoint of prosthetic reconstruction than the amputated leg. If this is true for a flail leg, it applies with still more force to cases in which some active muscles remain, as these can be made more useful than any mechanical device. He urges that every case be studied from this point of view before amputation is performed.

[ED. NOTE.—Little has found it necessary to issue such a warning as the foregoing. Surely, the knowledge should be spread to the four corners that, in a case of paralysis, amputation should rarely if ever be advised.]

Cinemaplastic Amputation.—Bosch Arana [92] describes two cases of cinemaplastic amputation of the forearm, in which he made a pincer-like stump by separating the radius and the ulna for a short distance, according to the method of Krukenberg and Putti. He found afterward, by study, that the closing of the two forearm prongs was due to the supinator brevis and the opening to the pronator radii teres. He states that the direct use of these fingers gives a very satisfactory function for holding small objects, and that their indirect use for motivating the artificial hand is of much value.

RESEARCH

Diseases of Blood Vascular System of Extremities.—Brooks [93] reports a series of experiments performed on dogs and rabbits, the object in view being the investigation of certain diseases of the blood vascular system of the extremities. In one series, the primary artery and vein of an extremity were ligated, both separately and together, and the effect on the tissue of the extremity was studied in detail. In another series, complete ligation was not done, but the effect of an alteration in the amount of blood circulating through the extremity was studied. The various effects produced by complete ligation and by variations in the amount of blood were analyzed, both in a general way and in their relation to the establishment of contractures, and the production of gangrene. An important observation in this series of experiments is that concerning so-called "ischemic paralysis" or "Volkmann's contracture."

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91. Little, E. M.: Brit. M. J., Feb. 16, 1924, p. 292.
92. Bosch Arana, G.: Chir. d. org. di movimento **8**:510 (June) 1924.
93. Brooks, B.: J. Bone & Joint Surg. **6**:326 (April) 1924.

[Ed. Note.—We feel that this work is of great importance, and that it cannot be properly abstracted. We feel also that great good will come from both reading and rereading this article in the original. The importance of understanding why Volkmann's paralysis or contracture is not an ischemic condition, and the importance of the observation made by Dr. Brooks on perivascular sympathectomy make of this experimental work a study of great practical value in surgery.]

Osseous Development in Endocrine Disorders.—Engelbach and McMahon [94] have made an extensive study of the osseous development in endocrine disorders. Their conclusions are as follows: The general diagnostic information derived from the roentgenologic comparison of endocrinopathic and normal subjects has led the writers to believe that the radiologic signs offer encouraging prospects of being of more value than the basal metabolism, blood chemistry and other so-called specific and laboratory determinations. Retardation of development of all the bones, not only of the carpals, in uncomplicated hypothyroidism can be demonstrated roentgenogically in all ages up to that of completion of normal skeletal growth. This will be an additional aid in those cases already beyond the age of normal carpal development. Hypogonadism and eunuchoidism have consistently shown a definite late fusion of the epiphyseal ends of the long bones. While this has been suspected clinically, we are unacquainted with any definite roentgenologic demonstration of these abnormalities in secondary hypogonadism. The late closure of the epiphyseal ends in the presence of an active hormone from the anterior lobe of the hypophysis explains the overgrowth of the long bones in these subjects. In the anterior lobe, pituitary insufficiency and a secondary deficiency of the generative organs, there has been found uniformly present a late closure of the epiphyseal ends of the long bones, associated with undergrowth of these bones. The reason for the undergrowth of the long bones in the presence of the open epiphyseal ends in this disorder is the absence of the hormone from the anterior lobe of the hypophysis. In the pluriglandular syndrome, the development of the osseous system as demonstrated roentgenologically is very difficult to interpret. From the studies of cases, thus far, the impression is that the following facts obtain:

1. In the thyropituitary disorder, there is an advance of the carpal and long bone nuclei development over that of pure hypothyroidism unassociated with pituitary disorder.

2. In pituitary thyroidism, there is a retardation of the appearance of the osseous nuclei, as well as of fusion of the epiphyseal ends of

94. Engelbach, William; and McMahon, Alphonse: Radiology, June, 1924, p. 378.

the long bones, more marked than that in pure hypothyroidism or in the normal.

3. The markedly heterogeneous pictures presented in the multiglandular syndromes will depend on the sequence in which the various disorders were superimposed on each other. For this same reason, the combination of the same glandular disorders might present entirely different roentgenographic pictures of the osseous development at the same age, depending on the order in which the various glands may have become involved. In the less frequent but very instructive condition of pubertas praecox (suspected pinealism), the most unusual advancement in development of the bone nuclei and early fusion of the epiphyseal lines was found. The four cases studied confirmed the earlier belief relative to the effect of gonad hormone on the osseous growth and development, and were a convincing confirmation of the exactly opposite picture consistently present in the hypogonad. Thymolymphatism in the few cases studied apparently presented much the same osseous retardation as mild hypothyroidism. The osseous development in positive cases of enlarged thymus should be more thoroughly studied, with the view of clearing up the much mooted point of the relation of thymus function to osseous development.

Effect of Roentgen Rays on Bone Marrow.—Falconer, Morris and Ruggles [95] report a series of experiments made on dogs to discover the effect of the roentgen ray on the marrow of the bone. They are led to believe from their study that direct roentgen-ray radiation of the long bones of a dog, in both light and heavy dosage, does not appreciably stimulate or decrease the contained marrow cells. Small doses, however, over the spleen cause an increase in the cells of the marrow of the long bone, and there is an increase in the proportion of immature marrow cells. Roentgen-ray radiation over the spleen also produces a rise in the number of platelets in the peripheral circulation. The general condition of the dogs which were thus exposed to the roentgen ray showed improvement during the experiments. The hemoglobin and red blood cells increased, and no ill effects were noted as a result of the frequent bone marrow punctures.

Alimentary Equilibrium and Bone Nutrition.—Moriquand and Michel [96] have performed a long series of animal experiments on nutrition of bone as related to diet. They are led to believe that the nutrition of bone is maintained in the normal condition by the balanced diet, which, in

95. Falconer, E. F.; Morris, L. M., and Ruggles, H. E.: Am.J. Roentgenol. **11**:342 (April) 1924.

96. Moriquand, G., and Michel, P.: Paris méd., May 3, 1924, p. 427.

addition to the necessary amount of calories, contains amino-acids, mineral salts and the three essential vitamins. If there is a deficiency in the amino-acids or in the mineral salts, the general nutrition is not affected immediately, and bone nutrition may not be affected at all if there is, at the same time, a sufficient dose of the antiscorbutic element. When to this diet is added an osteodystrophic agent of slight activity, the nutritive balance of the bone is broken, and immediately signs of bony change appear. These signs are usually osteohemorrhagic in character. These hemorrhagic disturbances will disappear when the diet is balanced and the deficiency is removed, even in the presence of the osteodystrophic agent. The importance of the alimentary equilibrium in the maintenance of bone nutrition is shown also in those cases to which are given a balanced diet, but one deficient in vitamin C (the antiscorbutic substance). When this deficiency is complete, bone lesions appear quickly. When it is partial, the lesions appear later. When the deficiency is slight, they appear only when the equilibrium of the diet is disturbed in some other way. Provided the equilibrium is restored, even when the vitamin deficiency continues, the bony lesions will disappear. If the alimentary equilibrium is disturbed, large doses of lemon juice will, in most cases, protect but not prevent the appearance of bony lesions. From this study, it may be assumed that one way of preventing osteodystrophy or disturbance of the nutrition of bone may consist in giving a well balanced diet very rich in fresh fruit juices. Without this dietetic base, all medication seems valueless, even dangerous. The authors believe that they have previously demonstrated this to be true in the use of cod liver oil.

Bone Grafts in Cases of Loss of Substance.—Bazy and Poussard [97] report the results of a series of experiments on animals in which gaps in the bone were bridged with bone grafts. They found that when a graft of 2 cm. was placed in a gap 3 cm. long, it filled in the gap completely in time. If the graft was reduced in length to 1 cm., repair did not take place. If the graft fails to unite to either end of the gap in the bone, it seems to lose its power of resistance to the destructive forces of the body, and gradually disappears in exactly the same manner in which it disappears when implanted in the soft tissues of the body.

Bone Growth Studied in an Exostosis.—Leriche and Policard [98] believe that the whole phenomena of bone growth is well shown in the growth of a simple cartilaginous exostosis. In this instance, the origin

97. Bazy, L., and Poussard, R.: Bull. de l'Acad. de méd., Paris, March 18, 1924, p. 731.

98. Leriche, R., and Policard, A.: Presse méd. **31**:905 (Oct. 31) 1923.

of bone growth lies in the detachment and eccentric arrangement of a tiny portion of cartilage. New bone is formed in a peripheral direction, and does not add to the length of the bone. The exostosis ceases to grow at the same time that the epiphyses unite. The authors consider bone growth at the epiphyseal cartilage, and observe that there are several stages in the progress of ossification. First, there is an arrangement of cartilage cells in a column which distally shows a deposition of lime salts in their intercellular substance. Second, there is an ingrowth of capillaries with absorption of the intracellular substance, and, finally, the laying down of bony lacunae in the spaces thus formed. The three phases are thus: proliferation of cartilage, absorption and new bone formation. Growth is the natural result of this process. We have no knowledge of what influences govern this process nor what causes it to stop. In a general way, they believe that the process of growth is under the influence of certain endocrine glands, but the specific agency is not at all understood. The innermost layers of the epiphyseal cartilage are most remote from the blood vessels, and it is difficult to understand how any hormone might reach these cells to excite their initial proliferation. Subsequent changes in the cells might result from the stimulation of metabolism, as the cells are approached by the vascularization of the epiphysis.

Experimental Studies on the Healing of Articular Cartilage.—Axhausen [99] has made experimental studies of the healing of articular cartilage. He produced in animals fractures of the articular cartilage and of the bone. He noticed that the bone healed exactly in the same way as bone distant from a joint. The articular cartilage, however, shows a very poor tendency to heal, and Axhausen believes that free bodies are not caused by impression fractures of the bones, but by pathologic fractures of dead epiphyseal bone.

The Nutrition of Articular Cartilage and Its Method of Repair.—Ito [100] has studied the nutrition of articular cartilage and its method of repair by experiments on animals. He believes that the synovia nourishes loose cartilage, and that it may be assumed that it has some nutritive function for unloosened cartilages, even in the center of the joint far removed from vascular circulation. Blood is supplied at the edges of the cartilage and from the vascular layer of cancellous bone on which the cartilage rests. His studies of the repair of cartilage differ markedly in the observations made from those of many other observers. His belief is that an injury to articular cartilage will, in time, repair by

99. Axhausen, G.: Arch. f. klin. Chir. **124**:543, 1923.
100. Ito, K. L.: Brit. J. Surg. **12**:31 (July) 1924.

fibrous tissue, which ultimately undergoes a metaplastic change, returning in its character to normal cartilage, thus making a complete repair of the injury.

MISCELLANEOUS

Bone Changes in Recklinghausen's Neurofibromatosis.—Brooks and Lehman [101] have made a study of seven cases. These abnormalities of growth and irregularity of outline of the shafts of long bones included changes which, in the roentgenograms, appear as subperiosteal bone cysts. Scoliosis, varying in degree, was present in all the cases. Excessive growth in length of long bones was noted in two cases. In one case, also, there was a marked inequality in the length of the two femurs, but apparently the longer bone was normal. Irregularity in outline of bones was found in five cases, varying, from very slight irregularity of the periosteal and cortical structure, to large tumors projecting from the surface of the bone or embedded as cystlike cavities in the structure of the bone. All the changes observed can be explained on the basis of the involvement of the bone by the growth of the tumor tissue, which is characteristic of Recklinghausen's disease. These observations emphasize the fact that Recklinghausen's neurofibromatosis is a condition affecting bone as well as the skin and nerves. The fundamental process in the disease is one of tumor growth.

Kümmel-Verneuil Syndrome.—Roederer [102] has reviewed all the literature bearing on the syndrome described by Kümmel and Verneuil and known as traumatic spondylitis. From this and from his own personal experience with several cases, he concludes that the condition really exists and that no evidence of fracture can be found in roentgenograms made immediately after injury. This does not exclude the possibility of a bony injury, and it is probable that this is present, although not demonstrable. Indeed, several necropsies have been recorded in which definite evidence of traumatic disintegration of the vertebra was found, even though the patients were not incapacitated and had resumed their work. Gradual softening occurs as a part of repair, and crushing takes place under the influence of the body weight. Roederer suggests that traumatic spondylitis may play a part in the production of essential scoliosis in children.

BOOK REVIEWS

Haglund of Stockholm and a number of Austrian, German, Hungarian, Dutch, English, Roumanian, Danish and Swiss collaborators

101. Brooks, B., and Lehman, E. P.: Surg., Gynec. & Obst. **38**:587 (May) 1924.

102. Roederer, C.: Paris méd. **2**:78 (July 19) 1924.

announce the launching of a new journal, *Die Erkrankungen des Bewegungsapparates Klinisch-therapeutische Vierteljahrsschrift.* The purpose of the undertaking is to focus on the organs of movement the light which is shed by the different branches of medicine, such as surgery, internal medicine, pediatrics, neurology, urology, orthopedic surgery and physiotherapy. The journal is to be edited by Haglund, and published quarterly by the Vienna Institut fuer Wissenschaftliche Hilfsarbeit.

THE PRINCIPLES OF ORTHOPEDIC SURGERY FOR NURSES. By James Warren Sever, M.D., New York: MacMillan Company, 1924.

Sever has written a most useful book on orthopedic surgery for nurses. It does not attempt to be exhaustive, nor is it highly technical. Orthopedic surgeons have long felt the lack of knowledge among trained nurses, both of the common bone and joint lesions and of the fundamental principles of the restoration of function. This book should stimulate more comprehensive teaching of nurses along these lines, and serve as an excellent textbook for this needed instruction.

INTERNAL DERANGEMENTS OF THE KNEE JOINT, THEIR PATHOLOGY AND TREATMENT BY MODERN METHODS. By A. G. Timbrell Fisher, M.C., F.R.C.S. (England) New York: MacMillan Company, 1924.

We might almost go so far as to say that no one who essays to diagnose and treat internal derangements of the knee joint should continue to do so until he has read this book of Timbrell Fisher. The subject is handled in a most thorough and enlightening manner. Old methods are discussed and original and successful new methods are illustrated. The normal and morbid anatomy is most descriptive. Throughout the book there is evidence of exhaustive investigation by the judicial mind of a trained surgeon of wide experience. We cannot speak too highly of this valuable contribution to medical literature.

ANNALS OF ROENTGENOLOGY, VOLUME IV, NORMAL BONES AND JOINTS, ROENTGENOLOGICALLY CONSIDERED. By Isidore Cohn, M.D., F.A.C.S. With a Foreword by Rudolph Matas, M.D. New York: Paul B. Hoeber, 1924.

Cohn's text and atlas of normal bones and joints go far in filling a real want of the surgeon who treats joints or lesions in their neighborhood. It is difficult to remember the exact normal stage of epiphyseal ossification at different ages and hard to interpret roentgenologic findings without comparison with the normal, not always available on the opposite side. The text is clear and the illustrations excellent. The section on the albow amounts to a sound dissertation on the treat-

ment of the various complicated fractures and epiphyseal separations of this region, in which future function is so important. We hope that, in a future edition, Cohn will include plates illustrative of those abnormalities of epiphyseal growth, such as the Legg-Calvé-Perthé's disease, osteochondritis, coxae juvenilis, apophysitis of the os calcis, Osgood-Schlatter disease of the tibial tubercle, Köhler's disease and Madelung's deformity.

Rudolph Matas has written a most important and discerning foreword. We commend the book.

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TRANSACTIONS OF THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

SEVENTH ANNUAL MEETING, ROCHESTER, MINN.,

JUNE 5, 6 AND 7, 1924

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CHARLES H. MAYO, M.D.

ROCHESTER, MINN.

Mr. President and Members of the Association:

We are, indeed, all very glad to have the association of intrathoracic surgeons meet here. The association is no longer in its infancy; it has reached the age at least of childhood, and already stands out as having among its members the best surgeons in the United States. In the past, all surgeons operated on the thorax, but the operations were mostly the occasional removal of a rib. This work has now advanced into the field of major surgery, and there is no specialty that is more deserving of such a designation than that which this society represents.

The war was of great benefit to science in hastening its evolutionary progress. The work of conquering the air is something that was accomplished within a few years during the war, whereas, under natural conditions, with less demand for air service, it would have taken an unknown number of years. Also, it would have taken a long time to have accomplished what the necessities of war accomplished very rapidly in submarine warfare. Likewise, the necessities and exigencies of war brought about rapid advances in the domain of medicine. For the first time in any war, the medical profession had an opportunity not only to prevent disease, but also to care for disease properly, and their work made a great impression on the millions of observing young men of our country who went to war, and I believe that the medical profession now has a much higher standing among at least 90 per cent. of them. This means a great deal for the future of medicine, and, consequently, in the development of all specialties.

The opportunity of medical men to care for an enormous number of patients with diseases of the chest, following the influenza epidemics, and of those who received injuries of the chest afforded opportunities to observe the results of various methods of treatment. As a result, the cumbersome, bungling procedures in vogue before the war have been supplanted by comparatively simple ones, and thoracic surgery is now done in the open, as it were.

SPECIALIZATION IN SURGERY

WILLIAM J. MAYO, M.D.

ROCHESTER, MINN.

The new specialty of thoracic surgery, which your association represents, and the specialty of neurologic surgery offer, possibly, the best examples of what might be called the modern tendency in surgery.

My father, in the early days when Charlie and I were his assistants, did the surgical work for a wide expanse of new country. He also found time for some general practice, and he had a wider knowledge of the medical science of his day than we can hope to acquire of our time. One of his achievements was the removal of approximately forty ovarian cysts, none of which weighed less than 20 pounds (9 kg.); one weighed 126 pounds (57.2 kg.). In those days, the cysts were not recognized until they were large, and the surgeon was not justified in operating in such a case until the family, the friends, and the neighbors of the patient could see that unless the tumor was removed the patient would die.

Today, it is impossible for any one man to know more than a little of comparatively few things. For this reason, thoracic surgeons are allied with internists and laboratory workers to secure the knowledge necessary for the proper and needful surgery of the chest.

In reality, it was the war that brought this society into existence, because it was possible during that time to examine many chests that had been opened very suddenly. In this manner, much practical knowledge was obtained of what was actually going on inside the thorax.

I have watched the growth of specialism of various sorts; for example, Drs. Carman and Moore and their associates taking roentgenograms of the stomach, and then coming to the operating room to see exactly how the object itself appeared as compared with their plates. By persisting in this method day after day for many years, they have become almost uncannily skilful in interpreting plates. The same is true of the urologists; Drs. Braasch, Crenshaw and Bumpus compare the findings at operation with those obtained at the examining table.

Thus, in the specialty of surgery of the chest, a combination of all these methods has enabled you to reach the position that you have, and it is really remarkable that, in so short a time, you have been able to learn what was going on in what was formerly a concealed area. Likewise, with regard to the skull, I remember, and not so long ago either, that about the only thing talked of by neurologic specialists was how to get inside the skull, whether one would use some sort of chisel, or an electric saw, or what; what one was to do after the skull

was opened is a question that has come up only in the last comparatively few years. We have seen the growth of this specialty parallel the growth of thoracic surgery here in the Mayo Clinic. Today, it is not so much a question how to open the chest as what is to be done afterward.

The whole problem of specialization in surgery is one that must be thought about very seriously, and in setting aside surgery of the chest as a specialty the question arises, Who shall perform these operations? I was interested in Dr. Butler's query a moment ago as to what the medical schools were going to do about teaching thoracic surgery. I repeat the question, What are they going to do about it? The senior students of medical colleges are working to their full capacity now. Measures to turn them into all round specialists should not be attempted. Things have come to the point where the real surgeon or the surgical specialist of the future must get his knowledge and training as a postgraduate. Instead of having these young men watch a brain operation requiring about three hours, we should teach them the vital principles that our fathers knew, how to take care of emergencies, in which the question of time is the most important factor.

I remember well what Dr. Finney of Baltimore once said with regard to obstruction of the bowels: that it is more important to operate early than to operate well, and that, if he himself had an acute obstruction of the bowels, he would rather have the occasional operator operate on him at once than the best surgeon in the world after a few days. Students should be instructed with regard to emergencies that certain things must be done early, but I am not at all sure that they should be taught to be specialists in all fields, or that the public should be given any such idea. The mere possession of a diploma does not endow one with extraordinary knowledge on all possible medical subjects. We shall soon learn that what we must try to do is to turn out from our medical schools a common garden variety of surgeon, one who knows enough to make a diagnosis, and who is thoroughly capable of handling emergencies, and who will have the good judgment to keep his hands off the things that are not immediately necessary.

The results, seen in many hospitals, of the general practitioner doing surgical work on the basis of his diploma are really appalling, but I do not believe this will continue. I don't know how it will be changed except as we change it ourselves with the aid of public opinion. I think the public is gradually finding out about the general practitioner who is doing an enormous amount of surgery that he should not do. We shall have different grades of surgeons, including certain ones who are, we will say, generally well trained, but who, when they encounter a brain tumor or a lesion of the chest, will be glad to refer it to a specialist in that particular field, because there is no possibility of any one surgeon knowing what to do under all circumstances.

It is very inspiring to see a new society formed by friends of mine, Dr. Willy Meyer and so many other established surgeons who are taking up this new field with a view to advancing it. It is only as surgeons get inspiration, not only from such leaders as yourselves in your particular branch but also from leaders in all the branches, that, to a certain extent at least, they will realize the necessity of specializing.

This leads to another question, the question of the whole-time teacher. I have no objection to the whole-time man from the standpoint of remuneration; the objection is that the man who might be most useful in the community, we will say a professor of surgery in a medical school, is tied down so that he cannot be useful to the people of that community or to the people of his state or the profession. Limit his remuneration if you like, but make him useful to all the people, and don't consider that if that man is professor of surgery he must be a teaching drudge and spend his time teaching the things already known in surgery, but let him emulate Evarts Graham and Heuer and others here who are doing research work, leading out in certain fields, advancing medicine, and who also are on call for professional and consultation work, doing something for all the people. The time has come when men must stand out; those who are capable of doing fine work should not be prevented from doing it because they hold a certain position, and it should not be expected of them.

THE EVOLUTION OF THORACIC SURGERY AS A SPECIALTY*

CARL A. HEDBLOM, M.D.
ROCHESTER, MINN.

Perhaps the most outstanding feature in the development of medical practice during the last three decades is specialization. This is the logical outcome of the vast increase in our knowledge of disease and its treatment. Surgery emerged as a specialty after the epoch making discoveries of Pasteur and of Lister. Early in the aseptic era, the number of recognized surgical procedures was so limited that there was no thought of specialization. But as the number and variety of disease conditions found amenable to surgical treatment increased, and as the number of operations multiplied and the technic developed, there naturally emerged the specialization of the surgery of the eye, ear, nose and throat, of the genito-urinary organs, of the extremities, and of the central nervous system. The general surgeon, in greater or lesser measure, still lays claim to the whole field, but there has been a growing trend toward specialization in one or a few allied anatomic systems. The leaders and those who have contributed most to the development of surgery have been, and will be, found in increasing measure to be those who have limited themselves to a circumscribed field.

Thoracic surgery is perhaps the latest of the surgical specialties to evolve. The founder of this association, Willy Meyer, has spoken of it as the youngest and last child among surgical specialties.

A consideration of the scope and the raison d'être of any of the specialties mentioned will show that there exists in each certain specific conditions which constitute in themselves ample ground for setting them apart. There is also the more fundamental reason that intensive study and training in any one field is productive of better results and more rapid progress. Thoracic surgery is no exception to these statements; there are structural and functional relationships, diagnostic and technical difficulties, and problems in after care which constitute as ample grounds for specialization as are to be found in any other field. The thoracic organs are encompassed by a bony framework which makes them relatively inaccessible. The thoracic esophagus is perhaps the most inaccessible organ in the body. Besides containing the great vessels and important nerves, the thorax is the seat of two of the three vital organs that constitute the tripod of life. Many of the other organs

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* Presidential address.

* From the Section on Thoracic Surgery, Mayo Clinic.

can be entirely dispensed with. The function of the gastro-intestinal tract may be kept in abeyance for days, but that of the heart or lungs cannot be interfered with, even momentarily, without threatening life. The diagnostic difficulties are so great as to tax the combined efforts of the specially trained internist, roentgenologist, endoscopist and surgeon. The technical problems are in many respects baffling, and differ in important features from those of other regions. Special methods of diagnosis and treatment, involving the use of the bronchoscope, the esophagoscope, differential pressure and other similar methods, require expert knowledge and skill.

Such being the case, it seems pertinent to inquire the reason why the development of thoracic surgery as a specialty has been late and its progress lagging. It is, I believe, bound up in the special characteristics mentioned as constituting grounds for specialization. Because of the difficulties involved, operation has been resorted to late, and the results of surgical treatment have been such as are obtained in the treatment of chronic conditions elsewhere, with more or less irreparable damage. The relatively meager field has also discouraged specialization.

The greatest obstacle to the development of thoracic surgery has been the disturbance to the vital functions of respiration and circulation produced most typically when the normal pleural cavity is opened. It is an elementary physiologic fact that respiration is based on the so-called negative intrapleural tension. It must follow then that open pneumothorax, which equalizes the pressure inside and outside the lungs, stops respiration on the affected side. If the evidence brought forward, especially by Graham, is accepted, namely, that in the normal pleural cavity, the mediastinal structures are so mobile that pressure changes on one side are transmitted through it to the other side, then the respiratory mechanism on the opposite side must also be interfered with. The venous return, depending in part on the aspiratory action of the negative intrapleural tension, is also impeded by changes in intrathoracic tension produced. Regarding the general effect on the patient and the consequent risk involved, there still is much difference of opinion, owing to the fact that patients have been observed who have had wide openings in the pleural cavity, traumatic or operative, without serious untoward effects. Based on such observations, sweeping statements have been made, especially since the Great War, to the effect that one need not fear open pneumothorax, that the chest cavity can be widely opened and explored just as freely as the peritoneal cavity, and that it is wholly unnecessary to resort to special differential pressure appliances for the maintenance of respiration. It is also frequently stated or implied that further progress in thoracic surgery depends in large measure on the dispelling of this groundless fear of open surgical pneumothorax.

In my opinion, further advance depends rather on the recognition of the limitations imposed by the physiologic conditions in the thorax, and on the devising of practical means for overcoming them. It is true that under certain conditions, open, unilateral, surgical pneumothorax leads to no marked untoward consequences, but it by no means follows that such is always the case. In many cases in which it has been said that open surgical pneumothorax has produced no ill effects, various technical procedures, such as partial obturation of the opening or traction on the exposed lung, have counteracted the bad effect of open pneumothorax by aiding the other lung in maintaining respiration. It is, however, the vital capacity of the patient in relation to the size of the unilateral opening and the relative integrity of his circulatory system that determines the relative amount of hazard that the open pneumothorax involves. The wounded soldier got well, in spite of his surgical pneumothorax, thanks to his large reserve vital capacity and his undamaged heart. The sick soldier died when a small opening was made in his thorax to drain an acute streptococcus empyema because of a relatively low vital capacity and a weakened heart. Civil thoracic surgery has to do in large proportion with patients who have similar lowered vital capacity and impaired circulatory function. Surgical pneumothorax means an added operative risk, the magnitude of which is in proportion to the reduction in the patient's vital functions, and it means an increased incidence of postoperative complications. This statement is in nowise qualified by the fact that patients may survive surgical pneumothorax, or by the fact that the introduction of differential pressure anesthesia has not led to the rapid progress that was anticipated.

Differential pressure anesthesia is sound in principle, and its general acceptance will obviate the greatest limitation to intrathoracic surgery. Efficient differential pressure apparatus is now so much simplified that it is within the reach of all, and the normal pleural cavity should never be opened without it.

It may be said, therefore, that the vital importance of the thoracic organs, their relative inaccessibility, the limited and inexact knowledge of the pathologic conditions in the living in any given case, and, specifically, surgical pneumothorax have retarded the development of thoracic surgery. There may be added also, perhaps, the relative lack of interest in this field and consequent failure to attack its problems due to counterattractions of more rapidly developing, less difficult and more remunerative fields.

That thoracic surgery has developed slowly is far from saying that no progress has been made. We have added to our knowledge of pathology, especially in the living. The impetus to the rapid development of abdominal surgery came not from the terminal pathology studied at necropsy, but from the pathology of the living, the stage of

the disease in which it was still amenable to surgical treatment. This will, I believe, be increasingly true of thoracic surgery as we advance in our correlation of pathology and clinical findings. We know, and the profession at large is beginning to realize, that the typical clinical picture of pulmonary tuberculosis may be present in the nontuberculous pulmonary suppuration. We know that cough and abnormal sputum may be due to a variety of pathologic lesions, the differentiation of which is essential to rational and efficient treatment. We are beginning to realize that the slightest amount of dysphagia may indicate carcinoma of the esophagus, and when the profession at large realizes this, we shall make progress in the treatment of this condition.

Certain fundamental principles of treatment have been established. The importance of prophylaxis against inspiratory infection of the bronchi and lungs during and following operations under general anesthesia is recognized. The fact is established that a large open pneumothorax is to be avoided, especially in the presence of a mobile mediastinum. We have begun to appreciate the value of preliminary preparation of patients for operation, and are learning to select operations more suitable to the condition requiring treatment. Necropsy findings in unexpected fatalities following operation have brought home to us the important fact that a chronic process may have produced damage to vital organs far beyond any clinical manifestations, and have emphasized the safeguard of the operation in several stages in such cases. We have come to realize the necessity for rest in certain types of disease, for intelligent exercise in others, and for a diet containing a high caloric content in all cases of suppuration. We are learning the value of the vital capacity test, both as a measure of the patient's general condition, and as a criterion of the results following operation. We are beginning to realize that it is not only the cure that counts but also the cost it represents in impairment of structure and function.

Among methods of treatment that have developed during the last two decades may be mentioned regional and differential pressure anesthesia, pneumothorax, extrapleural collapse and phrenicotomy in unilateral pulmonary tuberculosis, endoscopy, fluoroscopy, lobectomy and extrapleural collapse in pulmonary suppuration, mediastinostomy and pericardiostomy. The closed method of draining empyema and irrigation with antiseptic solution have been revived, particularly following the introduction of surgical solution of chlorinated soda (Dakin's solution).

Of the newer studies which may lead to important developments, those of Cutler and of Allen for attacking stenosed cardiac valves, ganglionectomy for angina pectoris and Graham's cautery lobectomy are outstanding.

The military surgery of the Great War brought out some important methods of localization and extraction of foreign bodies in the thorax and the necessity of the combined thoracico-abdominal approach in case of injuries in the region of the diaphragm, and demonstrated a remarkable degree of tolerance of healthy young persons to extensive thoracic operations.

Even a brief and fragmentary review of the present status of treatment of specific disease conditions provides ample evidence of progress.

EMPYEMA

For purposes of treatment, we distinguish between the streptococcus and other varieties of pyogenic infections, and in turn distinguish both of these from the tuberculous. In the acute streptococcic type, aspiration, or the closed method of drainage, only is permissible. We learned this lesson at a grievous cost, and must not allow it to be forgotten. In the other pyogenic forms, we have the alternate open and closed methods of drainage. In my opinion, if combined with efficient irrigation, the closed method represents a great advance in treatment. It improves physiologic function by producing reexpansion of the collapsed lung, and by relieving the pressure of the mediastinum on the opposite lung. The open operation disregards physiologic function in that it leaves the lung in status quo, or collapsed, and the cavity under atmospheric pressure, which in case of a mobile mediastinum reduces the normal negative tension on the opposite side. The closed operation, which is best carried out with the patient undisturbed in his own bed, is clean, painless under efficient local and nerve block anesthesia, and does not produce shock. The open operation usually involves the ordeal of transferring the patient to the operating room; anesthesia is more difficult; there may be bleeding; the pus is usually spilled about, and the combined effect of moving the patient, the thoracotomy, the sudden evacuation of pus, and the open pneumothorax may produce serious shock in an already critically ill patient. The closed method followed by irrigation is more efficient as a method of drainage, for the removal of pus is not left to gravity, but the walls of the cavity are washed clean, and the necrotic material and fibrin are dissolved and carried away. Moreover, the bactericidal action of the fluid sterilizes the cavity, lessening absorption of toxic material and promoting healing. The open method without irrigation depends for the counteraction against pneumothorax, as has been aptly said, largely on a pus-soaked dressing, or on a great variety of valve devices, the number and variety of which suggest their inefficiency. If irrigation is used in connection with open drainage, various precautions are necessary to insure irrigation of all parts of the cavity, a condition which is fulfilled automatically by the closed method by simply

filling the cavity. In case of large bronchial fistulas, open drainage is necessary and may be the most practical, following exploration for foreign body, osteomyelitis or multilocular cavities.

One of the greatest recent advances in the treatment of chronic empyema is the use of surgical solution of chlorinated soda, both as a means of obliterating the cavity and, failing that, as a preoperative preparation. By its intelligent use, large cavities of long standing may be reduced in capacity from 50 to 90 per cent., or even more. In the meantime, the patient gains weight, and improves with a promptness and to a degree often quite extraordinary. The patient comes to operation in a relatively good condition; the field is practically sterile, and the resultant sacrifice of structure and limitation of function are reduced to a minimum. In the cases of massive collapse of long standing with fibrosis preventing reexpansion of the lung, the operation can be performed in stages, the irrigations being kept up in the meantime. By a combination of such methods, shock can be entirely eliminated, and the mortality due to all causes and including cases with huge cavities and those with extensive damage to vital organs from prolonged suppuration has been reduced to under 5 per cent. The obliteration of concomitant, large, multiple bronchial fistulas can usually be accomplished, in a clean field, by silver nitrate cautery or by simple plastic procedures. In a word, we can point with justifiable satisfaction to progress in the treatment of empyema.

PULMONARY SUPPURATION

Pulmonary suppuration, in many of its forms, continues to be one of the most baffling problems confronting the thoracic surgeon, but a long step forward has been taken in the increasingly accurate differentiation into pathologic types and the selection of methods of treatment suitable to each. The prevalent diversity of opinion as to treatment is evidence of a growing interest in the subject. In my opinion, various methods employed have their definite indication, and it remains to perfect such methods and define their indications. In pulmonary abscess, expectant treatment, bronchoscopic lavage, pneumothorax collapse, drainage, lobectomy and cautery extirpation have their advocates. No one form of treatment is applicable to all cases. Expectant treatment, including postural drainage, is indicated in cases of early acute abscess that show evidence of improvement during a few weeks' observation, or at least do not manifest a downward trend. But its failures are numerous, as evidenced by the large proportion of chronic cases with extensive multilocular cavities and secondary bronchiectasis observed. In the Mayo Clinic, two thirds of all such cases seen are of the chronic type. Pneumothorax collapse seems rational treatment in acute, centrally located abscess, and drainage in peripherally situated cavities. Centrally located

abscesses are difficult to drain through the chest wall, and the risk of pneumothorax is great; but, in peripherally situated abscess, the lung is usually adherent, preventing collapse, and if collapse is achieved, there is the risk of perforation of the abscess into the pleural cavity. To treat such an abscess expectantly is irrational. Lobectomy or extirpation by cautery seem needlessly radical in such cases, in view of a recently reported series of cases with the low mortality of from 6 to 10 per cent. In the chronic, multilocular bronchiectatic abscess, extirpation seems the only rational procedure. Partial cautery excision seems to promise a lower mortality and the maximum of cures among those who survive the operation.

The most discouraging feature in the consideration of the treatment of bronchiectasis is the relative frequency of bilateral involvement. Of 318 cases observed in the clinic, 28 per cent. were bilateral, and in 36 per cent. the distribution was uncertain. The majority of these cases were, however, probably unilateral, at the onset, which suggests that operability can be increased by early recognition and prompt treatment. Expectant treatment, postural drainage and bronchoscopic lavage in these cases would seem, on pathologic grounds, to be palliative only. Removal of a foreign body from the bronchus certainly is curative before the bronchi have become extensively infected. Lobectomy in these cases has yielded a high mortality. Graded extrapleural thoracoplasty in my experience has been without mortality, and has produced a high percentage of improvement, approximating a cure. Secondary lobectomy following thoracoplasty has been reported by Sauerbruch, in six cases, without mortality. Cautery lobectomy, because it does not have to take account of adhesions, may prove to be the additional treatment necessary for a complete cure, in the cases not sufficiently improved by thoracoplasty.

In cases of multiple abscesses, the combined abscess and bronchiectasis with associated pneumonitis, extrapleural thoracoplasty has resulted in a mortality approximating that of drainage in acute abscess, and a relatively low percentage of marked cures. It may prove a useful adjunct, but extirpation seems the only logical treatment in these cases. Because it can be performed in graded stages, extirpation by cautery seems of the greatest promise in these cases, if secondary hemorrhage does not prove too frequent and serious a complication.

PULMONARY TUBERCULOSIS

The principle of rest in the treatment of tuberculosis seems well established. In case of a tuberculous lung, rest is accomplished by pneumothorax, or in cases of pleural adhesions, by extrapleural collapse. This method is, I think, accepted by practically all members of this association. Pneumothorax collapse is also being carried out in an

increasing number of cases throughout the country, but none of us has had any large experience with extrapleural collapse. We are far behind our colleagues on the continent in this respect. Sauerbruch alone has reported over 500 cases. Just why the development of this operation should be tardy in this country is not clear, but such seems to be the case. I am, therefore, particularly glad that we were able to arrange for a symposium on the subject of collapse therapy in pulmonary tuberculosis at this meeting. I am confident it will serve as an impetus to the more rapid development of the surgical treatment of pulmonary tuberculosis.

MEDIASTINAL SUPPURATION

Mediastinal suppuration has been generally considered almost uniformly fatal. That it is amenable to efficient drainage was shown by Lerche who, a year ago, presented before this association a series of five cases with four recoveries. I have had three cases of massive suppurative mediastinitis following mediastinostomy for exploration of esophageal carcinoma. All the patients recovered following irrigation with surgical solution of chlorinated soda.

NEW GROWTHS

With the exception of lipomas and dermoids, practically all new growths of the thorax are potentially malignant. Perhaps the most common is chondroma, which is a malignant tumor in the sense that it recurs, metastasizes and kills. All forms of treatment of sarcoma have yielded characteristically unfavorable results, but the radical removal of the relatively benign types has brought about some cures that appear to be permanent. Very large growths can be completely removed with a minimum of shock by operating in several stages. The vascular, rapidly growing tumors of the mediastinum, particularly Hodgkin's disease and lymphosarcoma, are amenable to radiotherapy, which at least prolongs life and makes it more tolerable.

Tumors of the lung are amenable to early surgical extirpation. Lilienthal, in his address before this association last year, forcefully called attention to this neglected field. I have attempted radical operation in one case, but the growth was too extensive for complete removal. Keller has performed a cautery lobectomy in one case with remarkably good immediate results.

CARCINOMA OF THE THORACIC ESOPHAGUS AND CARDIA

Carcinomas of the thoracic esophagus and at the cardia have continued to baffle our best efforts, but enough has been accomplished to sustain hope that we shall yet be able to put early radical extirpation on a practical basis. In my opinion, early diagnosis is the first essential to

progress in this difficult field. Two recent developments seem of definite promise. One is the reintroduction of the right posterior extrapleural mediastinostomy which gives adequate access to the thoracic esophagus without the production of pneumothorax. The other is the demonstration of the fact that infection of the mediastinum, incident to operation, is by no means necessarily fatal. As already mentioned, I have had three cases with extensive purulent infection with recovery.

BENIGN LESIONS OF THE ESOPHAGUS

Much progress has been made in the treatment of benign lesions of the esophagus, particularly of cardiospasm by hydrostatic dilation, and of cicatricial strictures by the silk thread and bougie dilation technic. The results of such treatment are highly satisfactory; and in many instances of long standing, in cases in which the patient was reduced to skin and bones, the immediate improvement has been positively dramatic. Apparently hopeless cases of stricture with esophagobronchial fistulas have been cured following dilation of the stricture.

PERICARDIUM AND HEART

Aspiration of a serous pericardial effusion through a small needle at the left border of dulness is technically almost as simple, and probably almost as safe, as aspiration of a pleural effusion. If exploratory aspiration was resorted to more frequently, there would be a smaller proportion of cases diagnosed at necropsy. The relief of cardiac embarrassment in case of a large effusion is also urgently indicated. The result of open drainage of purulent effusions has been discouraging. This is probably due in large measure to the lack of efficient draining against gravity, particularly of the posterior culdesac. A closed method of drainage and irrigation, as in a case reported by Whittemore before this association two years ago, may prove the solution of this difficulty.

Approach to the heart by a combined laparotomy and pericardiotomy, cutting through its attachment to the diaphragm, may prove a valuable method in that it affords access to the posterior aspect of the heart and pericardium. The approach to the valves through the auricular appendage and the cardioscope of Allen may open up a field of intracardial surgery. Cardiolysis for adhesive pericarditis is a rational procedure which seems to have been neglected. In an advanced case with greatly enlarged and damaged heart, often decompensated, but little is to be expected, but the operation is in itself not formidable, and, if performed before the heart has become seriously damaged, should yield gratifying results.

There are a number of other surgical conditions, perhaps not commonly recognized as belonging in the domain of thoracic surgery, which, nevertheless, by virtue of anatomic relationships, logically belong

there. Among such conditions may be mentioned diaphragmatic hernia, subdiaphragmatic abscess, tumors of the stomach involving its cardiac portion, and diseases of the breast. The pneumothorax produced by laparotomy for a diaphragmatic hernia differs in no respect from that produced by an opening in the chest wall. Some types of diaphragmatic hernia require thoracotomy to effect reduction and closure. Congenital hernia, including large defects in the diaphragm, can be closed only after preliminary thoracoplastic collapse. The diagnosis of subdiaphragmatic abscess involves exclusion of pleuritis and other conditions about the diaphragm, and its drainage requires special technic for avoiding infection of the pleural cavity. Such abscesses may perforate into the pleural cavity or a bronchus. Complete gastrectomy for tumors of the cardiac portion of the stomach can be technically greatly facilitated by preliminary thoracoplasty. Heuer's suggestion, to transpose the diaphragm to a higher level on the esophagus to make possible secondary anastomosis, may prove a valuable method in suitable cases.

Tumors of the breast anatomically fall within the scope of thoracic surgery. Further study of the avenues by which a malignant growth spreads may lead to modifications of technic, such as resection of mediastinal glands, and in suitable cases to resection of involved portions of the chest wall. Even though such procedures only prolong life, as is now the case in a large proportion of palliative radical resections, it would seem worthy of consideration. There is also need for further study in the correlation of radiotherapy and surgery in the treatment of carcinoma of the breast.

Attempts to forecast future developments in thoracic surgery would be perhaps presumptuous and unprofitable. A consideration of lines of development that seem promising may, however, be worth while. There is need for propaganda directed toward prevention of aspiration infection of the lung in ordinary chronic processes by more prompt and efficient treatment of the acute processes, and for early diagnosis and extirpation of new growths. Prompt aspiration of serous effusion, possibly coupled with the administration of intravenous antiseptics, may help to prevent empyema. Much remains to be done in the study of the pathology; types of bacterial infection in pulmonary suppuration; problems involving the mechanism and nature of pleural shock; pleural effusion and absorption in normal conditions of the lung, in different degrees of pulmonary collapse, and types of infection, and the part played by impeded venous return in the disturbances associated with pneumothorax. There is need for more prompt and accurate diagnosis of common pulmonary diseases, and especially early diagnosis of malignant disease. The rank and file of the profession should have the benefit of our experience in these matters. Questions concerning treatment are many: the proper choice of known operations, the development of

new methods. A uniform method of producing pleural adhesions except by suture and by infections has not yet been found. Lobectomy is not yet a safe operation. Resection of the esophagus has still to be put on a practical basis. The extent and practicability of cardiac surgery remain a question. The results of treatment of pericarditis with purulent effusion have been unsatisfactory. Much remains to be done as to localization and standardization of operations for diaphragmatic hernia. The question of applicability of the cautery and electrothermy to thoracic surgery is just being raised. Properly conducted animal experimentation should contribute much to the solution of many of these problems.

CONCLUSIONS

Thoracic disease and its treatment present complex and difficult problems demanding the combined intensive study of the internist, the surgeon, the endoscopist, the roentgenologist and the experimental worker. Thoracic surgery involves special problems of a nature as difficult and complex as those of any other surgical specialty, and specialization in this field may as reasonably be expected to further and hasten progress as in any other. As a specialty, it is still in the process of evolution. No better evidence perhaps of this statement can be cited than the existence of this association. This field presents many very important and difficult problems, but sufficient progress has been made to give us confidence as to its future development. The American Association for Thoracic Surgery may be looked to with confidence to furnish much of the leadership and impetus to such progress.

THE FUTURE OF THORACIC SURGERY

WILLY MEYER, M.D.

NEW YORK

Our country, at present, is forging ahead in the rapid evolution of thoracic surgery, which, truly, is the last child of operative surgery. There is now no nook or corner in the body that is not open to the knife, and safely so. That is one of the sources of the great satisfaction we derive from working in thoracic surgery.

To look into the future is not an easy matter. I believe, and I am sure you agree with me, that thoracic surgery will have its evolution in the same way that abdominal surgery has had, only it will have this evolution still faster, because we are standing on the experience we have had in general surgery, particularly in abdominal surgery.

I have been thinking of what Dr. Butler put before us in his usual plain and modest way. The proposition he has made, namely, that thoracic surgery should be added to the education of the student in our universities and colleges, seems to me to be a splendid one. The student should be taught this, not so much to become a thoracic surgeon, but that he will be able to choose the correct treatment in cases of emergency. Just think of the many serious automobile accidents that occur daily. The patient with an injury to the chest is carried to the nearest hospital. There is no time for temporizing or calling in a specialist. On the spot, the decision has often to be made how best to try to save life. The doctors way down in Arizona should know the method as well as the one living here; and he ought to learn this in the medical college. For, unfortunately, not every student that has passed his examination can become a hospital intern. Therefore, sufficient thoracic surgery should be taught at the medical colleges to prepare the future doctor to steer rightly, if life depends on his immediate decision. I believe, that many, thus taught, working in smaller hospitals, will find the subject so fascinating, so conducive to continuous personal thought, that they will stick to it and gladly add it to their daily routine operative work.

You have heard from our chairman what has been done so far in thoracic surgery here and abroad. In many of its chapters, we have a clear conception today and know the proper and best advance. In others, we are still in the stage of evolution. Let us as surgeons always remember that there is no other region in our body in which more specialties join hands than within the thorax. Let us gladly ask for counsel whenever a thoracic case in any of its aspects puzzles us.

One chapter, in which I have been especially interested for many years, I should like to mention. It is the surgical treatment of cancer of the esophagus.

I shall never forget what Dr. Meltzer, our first president, said in one of the meetings of the New York Society for Thoracic Surgery, when the question came up whether these patients should be operated on or be turned over to the men who use radium. The late Dr. Henry H. Janeway of the General Memorial Hospital had just told us, when asked about his experience with radium in this class of cases, that so far he had thirty-five patients thus treated, but not one had recovered. Dr. Meltzer then said, "More of these patients should be referred to the surgeons for operation. How will the surgeons ever make progress if you deny them the material on which and with which to gain experience?" That is perfectly true. In a discussion at this meeting, it was stated that the pendulum is now swinging back to surgery. At the present moment, it seems to me, we have to try to influence the profession at large to appeal immediately to the surgeon in these cases, now that the specialists in radiotherapy have openly expressed their opinion that they cannot cure these patients.

It is also our duty to educate the public, which means principally the patient's relatives, to have the patient submit to an operation rather than to ray treatment, now that the outlook for real help by means of operation has brightened. If operative exploration proves the impossibility of a radical cure, then ray treatment (radium) is indicated. I believe the Association for the Control of Cancer can help surgeons immensely in this crusade by directing unfortunate persons afflicted with the disease properly and early.

And cancer of the cardia! Every surgeon is ready to operate for cancer of the pylorus, but the majority of patients afflicted with cardiac malignancy are left to die. Yet, the patients have the same right to hope for medical help as if the tumor had grown within another portion of the esophagus or at the pylorus. Here, too, surgery must become more aggressive, since we have learned how to avoid the dangers and how to proceed technically.

So, looking ahead, I believe the future of thoracic surgery is bright. I believe that every surgeon should add at least so much of it to his daily work that he can give proper and intelligent help to the thoracic patient in emergency.

THE SCOPE OF THORACIC SURGERY*

AMBROSE L. LOCKWOOD, M.D.

TORONTO

From 1915 to 1920, as a result of the war and the dreadful epidemic of influenza, thoracic surgery developed to a greater extent than it had in all previous time. A sufficient period has now elapsed for those of us who have been more closely interested in observing these patients to have arrived at certain definite conclusions as to the merits of the methods of treatment employed, the results obtained, and the future scope and possibilities of thoracic surgery generally.

This society should be the proper judicial body to decide and broadcast the standard methods of diagnosis, treatment, prognosis and aftercare of diseases of the thorax and its contents, including affections of the chest wall, the lungs, the heart and pericardium, the mediastinum and its contents, the diaphragm, and diseases of the organs adjacent to the latter demanding a thoracic abdominal approach.

It is from a desire to stimulate discussion in the society on these problems generally that I have been prompted to select so vast a subject.

It is impossible, in this presentation, to deal at length with the various affections of the thorax and its contents, but I wish to touch briefly on the more common conditions.

EMPYEMA—ACUTE AND CHRONIC

Even the youngest members of the society can recall the great dread with which we observed the development of an acute empyema following a pneumonia, the alarming febrile reaction, the grave consultations as to the advisability and time for operative intervention, the heavy mortality, and the large percentage of chronic empyema resulting, with the long drawn out convalescence, the distressing sequelae and the repeated operations necessary for the final obliteration or closure of the cavity in the chronic stage of the disease. As a result of closer study of empyema in general, measures have been established which have reduced our mortality in acute empyema to almost nil, the incidence of chronic empyema to a very low mark, and the total death rate to approximately 2 per cent. Mutilating operations, such as the Schede and Estlander, are rarely necessary and reexpansion and complete function of the affected lung can be obtained in the vast majority of patients. It should still be impressed on the profession that aspiration in the developing empyema should not be too early undertaken, and

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then only with the greatest care, to avoid collapse of a lung that is not as yet sufficiently firmly adherent to the chest wall. One should be guided by the elevation of temperature, pulse and respiratory rate, the degree of dyspnea, the displacement of the heart, and the toxic condition of the patient generally and aspiration should be delayed until the empyema is well walled off, and the lung about it firmly adherent to the chest wall, so that if air does enter the cavity, or if the walls of the cavity tend to collapse, as the fluid is aspirated, the adherent parietal and visceral pleurae do not separate, and a localized empyema cavity, which was pocketed and could easily have been dealt with, become transformed into a general empyema, occupying a large cavity in the chest, with its attending added area for absorption and problem of obliteration. Undoubtedly, many patients who have had a small self limiting pocket of pus which would have continued to wall off and absorb or perhaps rupture into a bronchus and be spat up, have, by too early or careless aspiration, developed an extensive empyema with all its dreadful sequelae. It is only in obscure cases that the aspirating needle should be necessary for diagnosis, and it should be borne in mind that the fluid may be a protective agent of nature, our greatest healer. Apart from the straw colored fluid found in tuberculous pleurisy, if the fluid in the syringe is clear and flows freely, we have aspirated too early, and except to relieve dyspnea, no more than a few ounces of fluid should be taken off at that time. It is astounding and inexplicable how rapidly absorption of even fairly thick fluid occurs within the thorax, and no one can state how much, or how little, or how soon, or how late absorption may occur.

In all our treatment of empyema it should be borne in mind that the fluid is first a protection, to splint the lung and stabilize the mediastinum, and that the early deaths are not due to absorption of the fluid per se, but in the majority of patients, to too early intervention with the fluid, collapse of the small remaining air bearing portion of lung tissue, and insufficient oxygenation. Death results not from the disease itself, but from injudicious intervention by the medical or surgical attendant.

We must look on chronic empyema as an improperly treated acute empyema, and I agree with Whittemore, who aptly put it, "that the cause for an acute empyema becoming chronic is due to one of three reasons: either it is a neglected case and has not received surgical attention when it should have, or it has been badly operated on, or the after-care has been poorly managed."

Radical operation should be undertaken only after the exact size, nature and accurate ramifications of the cavity have been determined, and when, after a thorough trial of the Dakin treatment, the application

of various dyes, blowing exercises and suction, etc., the cavity is no longer obliterating. Operation should not be delayed, however, once the cavity has reduced as much as it is apparently going to with palliative measures, and once the patient's general condition has improved as much as we can expect it to, with the thorax still draining.

In all cavities which contain more than 3 ounces (89 c.c.), effort should be made to reexpand the lung to occupy the cavity. In most of the cavities under 3 ounces (89 c.c.), especially if peripherally placed, closure by muscle or skin flap, moderate resection of the chest wall, or some allied method is advisable.

In patients with the larger cavities, there is a diminution in the vital capacity of the lungs and every effort should be made to expand the collapsed portion, thereby restoring the air bearing capacity of the lungs, and, as well, obliterating the cavity. Decortication properly performed, when followed by complete expansion of the collapsed portion of the lung, is one of the most satisfactory operations in modern surgery. There is a certain percentage of patients in whom a fibrosis has developed in the collapsed lung and in whom expansion is thereby limited, but it is rare that some expansion does not occur, and it has been quite impossible, for me at least, to prognosticate whether or not the lung will expand after I have completed the decortication. However, I have now, after many painful experiences, so modified my method of decortication that I no longer have any ill effects from the procedure, even though the exposed lung does not expand. Decortication in inexperienced hands, or in the hands of the general surgeon who only occasionally is called on to deal with a patient requiring such an operation, is an extremely difficult procedure, fraught with many dangerous possibilities and sequelae.

Such operations as a modified Estlander and Schede operation are unfortunately still necessary in a certain percentage of patients. With our present technic, however, the complications and dangers of such mutilating procedures have been reduced to a minimum.

All of us have seen large numbers of patients suffering from chronic empyema who have gone on for months or years still draining, and we have surprised ourselves by the ease and dispatch with which we were able to obliterate the cavity and return these patients to health. The location of the opening in the chest or the amount of discharge is no indication of the size or exact location of the cavity. In every community, there are patients with chronic empyema who consider themselves incurable. It is the duty of the members of this society to educate the profession generally, and even the laity, to the possibility of benefiting all, and completely curing most of these patients without much morbidity, and with almost no mortality.

LUNG ABSCESS

Fewer patients suffering from this disease now require surgical treatment, but when the operation is necessary it is more radical and logical, and results in a higher percentage of cures. The actual mortality in patients operated on may be higher than heretofore because they are of a more extensive and resistant type and have not responded to medical treatment, but the combined mortality in patients suffering from lung abscess treated medically and surgically has been very much reduced. Postural drainage, rest in the open air, forced feeding, artificial pneumothorax and bronchoscopy will effect a cure in most patients. Operative procedure should only be undertaken after the former treatments have failed. Operation should not be delayed, however, until the patient has developed albuminuria, arthritis, myocarditis and amyloid disease. When the patient is no longer improving or holding his own under palliative treatment, operation should be done.

There are few conditions which, arising often from such trivial and preventable causes as does abscess of the lung, so quickly and completely incapacitate a person, rendering him a burden to himself, his friends and society. The progressive weakness, the distressing cough, the long convalescence, and, above all, the foul smelling and tasting sputum break the morale of the stoutest patient and present one of the most difficult conditions to relieve.

It is unnecessary to lay stress on the incidence of lung abscess following operations on the nose, throat and buccal cavity under general anesthesia, and particularly after tonsillectomy. Admitting and fully realizing, as we must, the danger of tonsillectomy under general anesthesia, and the comparative rare incidence of this disastrous complication when local anesthesia is employed, in view also of the ease of performing tonsillectomy under local anesthesia, with due respect to our associates practicing otolaryngology, the time is overdue for members of this society to go on record as opposed to the employment of general anesthesia for operations about the nose, throat and buccal cavity, and more particularly for tonsillectomy, when the operation can as readily and so much more safely be performed under local anesthesia. Otolaryngologists, the profession generally, and, if necessary, the laity should be thoroughly aroused to the danger of lung abscess after tonsillectomy under general anesthesia.

BRONCHIECTASIS

Unfortunately, patients with bronchiectasis rarely come for consultation early enough for proper surgical treatment, and only after thorough medical treatment along the lines advocated for lung abscess should surgery be instituted. Partial pneumectomy is advisable in

limited types of unilateral bronchiectasis and artificial pneumothorax or surgical collapse in a certain percentage of cases. Graham's suggestion of pneumectomy by repeated cauterization may prove of value.

I am about persuaded to resort to diathermy in extensive unilateral bronchiectasis. In bilateral bronchiectasis, I still feel that collapse of the more severely affected lung is the limit of judicious surgery. The best result in such patients is partial rather than complete cure.

FOREIGN BODIES

Foreign bodies can be safely removed almost regardless of where they are lodged within the thorax. Particularly in the mediastinum and lung, an embedded foreign body is usually surrounded by a limited degree of infection, and is a potential focus. Localized sclerosis about a piece of metal in the lung spreads to a diffuse sclerosis in a certain percentage of cases. Although many of these patients have no cough and do not produce sputum, they have recurrent attacks of fever, chills, general malaise and acute rheumatism. The majority of such patients worry about the presence of the foreign body in what, to their minds, is a dangerous location. After having refused to operate on many of these patients at first, and after long observation of them, I have been forced to the conclusion that they should be dealt with by judicious surgery.

TUMORS OF THE THORACIC WALL

Tumors of the thoracic wall should be attacked surgically as soon as diagnosed, provided they have not developed metastasis or spread so widely that even, after eradicating the primary growth, fair promise of extending life can be assured. Resection of the underlying attached lung or pericardium must be fearlessly resorted to. Radium and deep roentgen-ray therapy should be pushed to the limit. Emile Beck has pointed the way in these otherwise hopeless conditions. While we cannot hope for much from carcinoma, we do not yet know what to expect from such treatment of sarcoma.

INTRATHORACIC TUMORS

It is unfortunate that more patients suffering from intrathoracic tumor do not come to surgery sufficiently early to warrant operative intervention. I have been forced to the conclusion that where doubt exists as to the presence of a primary carcinoma, or as to the nature of a demonstrable growth, rather than delay or apply dangerous palliative treatment, such as roentgen ray, exploration and quick section of a portion of the growth should be resorted to at once. I have regretted having delayed operation in several cases, and I have never seen the least harm done by an early exploratory operation. Roentgen-ray

therapy has undoubtedly hastened the growth in several primary carcinomas of the lung which I have seen. I have not yet seen any benefit follow roentgen-ray therapy of intrathoracic carcinoma. Early primary carcinoma or sarcoma within the thorax should be cured by radical excision, followed by radium and roentgen-ray therapy, almost as readily as elsewhere in the body. I believe that every adult over 35 years of age should have stereoscopic roentgenograms made of the thorax, whether or not he has definite thoracic complaints, before he is given a clear bill of health. The expense is not great and only in this way will we pick up early growths within the thorax.

Of course if there are definite metastases, or if there is fluid in the thoracic cavity, or if when the thorax is opened the mediastinal glands show metastasis, further surgery, except for relief of symptoms, in contraindicated.

I have decided that, in a widespread, peripherally placed carcinoma of the lung, or in a circumscribed growth that cannot be readily exposed and freed, I will resort to direct diathermy of the growth and of a wide area of the surrounding lung tissue. By this method, one should be able to eradicate the disease completely with the least possible risk to the patient. In animals, at least, it is remarkable how easily and safely resection of lung tissue can be carried out by diathermy without any bleeding or shock whatever. I should appreciate criticism of this suggested method of excision.

CARCINOMA OF THE ESOPHAGUS

I must confess that I am pessimistic about the possibility of radical cure of carcinoma of the esophagus. The patients come to surgery so late that so far we have not accomplished much. However, we should persist in our efforts to perfect some method of dealing with this dreadful condition. A certain percentage of patients with carcinoma of the esophagus, diagnosed early, may yet be cured by excision through a thoracic approach. Drs. Torek, Meyer, Lilienthal and Hedblom deserve the greatest credit for their effort to perfect some method of dealing with this condition.

PURULENT PERICARDITIS

In patients with purulent pericarditis, unless the pus can be aspirated readily and the patient thereby improved, operation should be done early. An ample flap of the chest wall should be turned back to the left of the sternum, the pericardium opened widely and the pericardial sac thoroughly swabbed out, the operator, being careful to clear out the pus posteriorly where it tends to pocket in the pouch over the transverse sinus. Pus in this pocket is difficult to aspirate except posteriorly, a dangerous and uncertain approach.

SUBDIAPHRAGMATIC ABSCESS

Subdiaphragmatic abscess and high or deeply placed hepatic abscesses can best be approached through an extensive abdominothoracic incision. Operation should not be done too early in these cases. I have recently seen a patient with double subphrenic abscess following a pelvic cellulitis who was moribund. Operation could not be undertaken without the greatest risk to the patient. With rest in bed in the open air, forced feeding, fluid by rectum, and heat to the entire abdomen, the abscess on the left side first ruptured into the stomach and was vomited up, and later the one on the right side burst through the diaphragm and was spat up. Postural drainage was instituted, and the patient entirely recovered without any operative intervention whatever, returning to her home seven weeks after coming under our care. This is a rare result; however, it serves to illustrate the value of conservative treatment and close observation of patients when more radical treatment cannot be carried out without too great risk.

HEART

Many wounds of the heart were dealt with during the war. With care in manipulating the organ so as not to kink the vessels at its base, the heart can be freely handled and lacerations repaired. Great credit is due to Dr. Elliot Cutler for his recent work on the valves of the heart. The practical value of his work remains to be seen, but his daring effort to relieve stenosis of the valves should be applauded.

SURGICAL TREATMENT OF UNILATERAL TUBERCULOSIS

Dr. Archibald deserves the greatest credit for his contribution to surgical treatment of unilateral tuberculosis. There is a type of unilateral pulmonary tuberculosis in which artificial pneumothorax has failed to collapse the lung that demands surgical attention. The type of patient requiring such treatment should be carefully selected by the internist specializing in tuberculosis, working in the closest harmony with the thoracic surgeon. Excision of portions of the ribs posteriorly and laterally in two or three stage operations gives the best promise of benefit with the least risk to the patient. Properly selected patients, in the hands of a surgeon experienced in thoracic work, operated on under a selected anesthesia, can be greatly benefited, with scarcely no mortality rate.

TRAUMATIC LESIONS OF THE THORAX AND ITS CONTENTS

I have left the problems of traumatic lesions of the thorax and its contents to the last, and I regret that time will not allow me to go into this subject at length. I propose to make a more lengthy

contribution to it shortly. It was definitely established, during the war, that many deaths were due not to direct injury of vital organs per se within the thorax, but to the shock and exhaustion caused by the gross, bony lesion of the thoracic wall and the distressing respiratory movements caused by the visceral pleura coming in contact with the comminuted bone and sharp, bony spicules. It was only by active resuscitory measures, by early operation and removal of the overriding bone and sharp spicules that we were able to save the lives of many of these patients.

I am now of the opinion that many of the deaths following automobile accidents, train wrecks and amongst men injured in construction work are due to what I have previously described as a stove-in chest. The great majority of men injured in this way could be saved if they were dealt with properly. Shock should be combated by every possible means. Morphin should be freely administered. They should be placed in the most comfortable position to offset dyspnea. If possible, stereoscopic roentgenograms of the chest should be taken with the minimum of disturbance to the patient. If the pain is intense over a fairly localized area of the chest wall, paravertebral and local block anesthesia should be at once resorted to to relieve it. A blood transfusion should be given if there is a large hemothorax or if much blood has been lost otherwise. Operation under paravertebral and local block anesthesia should be undertaken when the patient's condition will permit.

Stab wounds and gunshot wounds of the chest should be dealt with promptly. Apart from wounds of large vessels or of the main bronchi causing death almost at once, death in these patients reflects on the attending surgeon. No man should ever die from a progressive hemothorax. I have been watching reports on these types of wounds, and deaths are still occurring in a type of wound which is not fatal if properly dealt with. In every large center, there should be one or two men who have had special training in thoracic surgery, and these men should be called to deal with thoracic injuries. Easily avoidable deaths are occurring almost daily in all our large cities from thoracic injuries which could be avoided if the general surgeon turned these patients over at once to men who have been trained to deal with them. I will defy death in man or beast following a gunshot wound or stab in the thorax if death is not instantaneous from laceration of the main vessels, auricles of the heart, or severance of the main bronchus, provided I can get the wounded within from one-half to two hours of the receiving of the lesion. The wound in the chest wall should be firmly closed with deep silkworm gut sutures, or sealed over with adhesive plaster; morphin should be given, the patient placed in the most comfortable position to offset dyspnea, and a thoracic surgeon should be called in consultation.

Laceration of the diaphragm causes a variable degree of distress. Such a lesion can usually be readily diagnosed and can easily be repaired through the thoracic route, unless there is a complicating abdominal lesion which demands attention at the same time and the diaphragm can be approached through the same incision.

DIAGNOSIS OF THORACIC CONDITIONS

An internist specializing in thoracic work, a roentgenologist particularly interested in thoracic lesions, a careful anesthesist thoroughly experienced in gas-oxygen administration, and a general surgeon who has had long experience in thoracic surgery, should be closely associated in the diagnosis, prognosis and treatment of thoracic lesions.

Full-sized stereoscopic roentgenograms of the thorax should be made. Movements of the thoracic contents and diaphragm can be determined only with the fluoroscope. The roentgenograms show the approximate amount of fluid, the presence of pneumothorax, the degree of collapse of the lungs, the position of the heart and mediastinum, the level of the diaphragm, the extent of bronchiectasis, the cavitation of the lungs, the presence of active or quiescent tuberculosis, metastasis, hematoma of the lung, the presence of foreign bodies and the position, size and contour of a mass. Repeated roentgenographic examinations are necessary during the progress of the case.

CHOICE OF ANESTHESIA

With all due respect to the members of this society who still continue to employ general anesthesia in their thoracic surgery, without any apology or temerity, I still maintain that paravertebral anesthesia, associated with a block anesthesia and gas and oxygen analgesia, if necessary, should be the anesthetic of choice in all our thoracic work.

I can see no reason whatever why, if one lung is partially or completely incapacitated, we should employ any anesthetic which might endanger the air-bearing capacity of the other lung. Furthermore, a more deliberate and protracted operation can be carried out under local anesthesia with less risk to the patient. The cooperation of the patient in holding his breath or forcing out his lung greatly facilitates the operation. Time will not permit me to deal at length with the question of anesthesia, a subject which I have already harped on considerably. Suffice it to say, however, that I have employed paravertebral anesthesia in nearly 3,500 patients. I have never had a death, nor anything approaching a dangerous sequela, that could, in any way, be attributed to the anesthetic. I never allow a patient to suffer either mentally or otherwise. By the proper use of a preliminary narcotic and gas and oxygen analgesia, if necessary, the most nervous type of patient can

be operated on without the least anxiety. I never inject the solution into the intervertebral foramen, but catch the nerve under the lower border of the rib at its angle posteriorly, employing 1 per cent. procain for the paravertebral injection and 0.5 per cent. for the local infiltration. I have operated on many patients, under this type of anesthesia, who could not possibly be given a general anesthesia, and I have not yet had a patient show any alarming ill effect from the amount of procain employed. The present modified method of paravertebral anesthesia is simple, almost fool proof, and I must confess I cannot understand why it is not universally employed in thoracic surgery.

In conclusion, the members of this society have reason to be proud of the success of our efforts in this new field of surgery, when due credit has been given such men as Sauerbruch, Tuffier, McKewan, Duval, Gaske and others in Europe, it is largely through the constructive efforts of members of this society that thoracic surgery has developed to the high degree of perfection now attained.

We can point to the greatly reduced mortality in acute empyema, to the very small percentage of chronic empyemas now being seen, to the smaller number of lung abscesses now occurring, and to the lessened death rate and morbidity from this dreadful malady, to the earlier recognition of the malignancy of the thorax and its contents, and to the more logical and thorough treatment of such disease, to greater benefit in the treatment of bronchiectasis, purulent pericarditis and mediastinitis, and to the great advances in the use of the bronchoscope as a diagnostic and therapeutic instrument. The surgeon members of the society owe a great debt to fellow members such as Jackson, Yan Kaner, Plummer, the late Dr. Lynah and many others who have developed bronchoscopy to such a high degree. What the cystoscope is to the urological surgeon, the bronchoscope is to the thoracic surgeon.

While the advances of thoracic surgery are widely recognized, extensive visits to the various medical and surgical centers of the continent have made me wonder whether or not the profession generally realize the necessity for the greatest care in the prevention of diseases of the thorax and its contents, the meticulous established diagnostic measures necessary, the close cooperation demanded between the surgeon, the internist, the roentgenographer and the laboratory man in determining the exact nature and extent of the lesion, the treatment to be employed and the after-care to be instituted.

DISCUSSION

DR. WYMAN WHITTEMORE, Boston: I agree with what Dr. Lockwood has said about the use of local anesthesia. I think it has been many years since I have used anything except local anesthesia; rarely, it has been supplemented with gas-oxygen. There has never been the slightest difficulty in operating on tuberculous patients for removal of nine or ten ribs with local anesthesia

I want to speak of a small group of chronic lung abscesses. This group consists of a few patients that have had a lung abscess over a long period of time, say from eight to nine months to two or three years, in which the abscess persists, the cavity, of course, being surrounded with some pneumonitis. Now when these patients have been drained, in my experience, they may fall into one of three different classes: First, those that die, second, those that do well as long as the drainage is kept up. They return to their work and do live a useful life. But the third group is the one that I am particularly interested in; these are the patients that do well following the operation in the hospital, go home and do well for two or three months, then begin to go down hill; they still have a small drainage tube in. They begin to have more cough and expectoration. This gradually becomes blood tinged; then there are small hemorrhages and then large hemorrhages until the patients finally die. They are most distressing cases. Drainage in these cases was not satisfactory.

This winter I have had two cases falling into this group of chronic cases, both situated in the upper lobe, both posttonsillectomy cases. It occurred to me that it would be possible to collapse the abscess, possibly draining it, but it did not seem to me possible to drain it at the same time as collapsing it; so, in each case, I did an extrapleural thoracoplasty operation, resecting a portion of the second and third ribs, pushing the apex of the lung well down, extrapleurally, and filling in the cavity with a flap of the pectoralis major muscle. It was suprising to me to see how well these patients did.

The first case was that of a woman who had had an abscess for eight or nine months. Within a week or ten days, her temperature had come down to normal, and it stayed there. Her cough gradually let up, and when she left the hospital, at the end of four or five weeks, she had no cough and no sputum. She has been followed for four or five months, and at the present time she still has no cough and no sputum, and she has changed from being a thin, emaciated woman into a fat, robust woman.

The other patient was a small boy 12 or 13 years old who had an abscess following tonsillectomy. I couldn't get up the courage to drain it, so I did the same operation. His convalescence in the hospital was very much the same. When he left the hospital, he had practically quit coughing, and had had a normal temperature for three or four weeks. He also has been followed. At the present moment, he has practically no cough. His mother says that once in a while, he coughs a little, but that what he raises is not foul at all, and the foul odor to his breath has entirely gone.

Of course, I realize that the two cases are nothing to base any judgment on, but the result in these two cases happens to be so good that I thought it worth while to speak of them.

DR. ETHEN F. BUTLER, Sayre, Pa.: Thoracic surgery has not yet attained the established position that abdominal surgery has won for itself. I therefore agree with Dr. Lockwood that this society might well interest itself in communicating to the general profession and to the laity such information as it has at its command, as to what may or may not properly be expected from surgical intervention in proper cases.

The action, taken two years ago, of having the transactions printed in the ARCHIVES OF SURGERY was a wise step in that direction. The next step might well be the peaceful invasion of the medical schools with the object of securing their cooperation. If the oncoming generation of physicians were

taught during their impressionable years the scope and possibilities of thoracic surgery, there would, in the future, be less popular and professional prejudice against its employment.

Dr. Willy Meyer, New York: Dr. Lockwood mentioned the paravertebral advance for local block anesthesia. I would call a warning with reference to that method. Two deaths have been reported from abroad, and I believe I have lost one case of extrapleural thoracoplasty in consequence of it. I had used this method of Kappis a number of times without any bad results. I believe the method of preparatory injection of procain, at the angle of the rib and during the operation, directly into the thoracic nerves is preferable.

Dr. Lockwood's survey with reference to thoracic surgery naturally covers a large field. I merely want to add the following: I just received a letter from Professor Trendelenburg, formerly of Leipzic, in which he told me that the first case of successful operation for pulmonary embolism had just been presented before this year's meeting of the German Surgical Congress in Berlin. He added that he is happy that he lived his eighty years to see his operation having saved a human life. Professor Kirschner of the University of Königsberg was the operator, and he followed absolutely the rules worked out and laid down by Trendelenburg.

We know that this operation was done repeatedly by Trendelenburg and his pupils before the World War. One patient lived five days after the operation and then died of pneumonia. Kirschner's case is the first that has been saved by aggressive thoracic surgery. It represents another leaf in Trendelenburg's laurel wreath.

Dr. Edwards S. Wells, Saranac Lake, N. Y.: I should like to add one word to what Dr. Meyer has said about paravertebral anesthesia, and in relation to gas. My work has all been with tuberculosis, and I have had a very bad impression of paravertebral anesthesia as it is carried out through the entire chest wall. I have come to the belief that it is more time consuming than is necessary. It seems to take so much time to get the long needle down through the whole thickness of the chest wall and the muscles and into the exact spot where it needs to be to give the complete anesthesia that I feel that it is very much faster and more satisfactory to put the procain in as I go down. I infiltrate the outer chest wall, cut through it, expose the ribs, get right down to the intercostal nerves and put the anesthetic in the nerves when they are under my finger; the procain injected at this time takes effect quickly enough to let me work on the ribs without waiting.

I think that there are cases in which a great deal of advantage can be gained by using a combination of local and gas and oxygen from the start. I have operated on some patients under local anesthesia only when I thought the effect on the patient mentally was worse than if I had used the combination, because local anesthesia has to be absolutely complete, and sometimes one has to stop in the middle of his operation when he hasn't secured complete anesthesia and put in more procain. It prolongs the operation and if the patient is of a nervous type, in spite of considerable premedication, I think it subjects him to a hard mental strain to go through such an operation during which he has to be in a pretty uncomfortable position on the table; I think these patients frequently do better if given a sufficient nitrous oxid analgesia to make it possible to use less complete local anesthesia. One can work right ahead and get the operation done quickly, and save time and mental strain on the patient. When there is a large amount of sputum being raised in the tuberculous cases, I think it is important to use a local anesthetic only, and that any general

anesthetic, even light nitrous oxid, seems to be somewhat dangerous; but in the case in which I have operated in which the amount of sputum was not large and the cavities not extensive, I have had no bad result whatever from using nitrous oxid throughout, but a very great advantage in time and in saving strain on the patient.

Dr. A L. Lockwood, Toronto: As regards the question of paravertebral anesthesia, since 1913 I have not injected into the intervertebral foramen. At that time, I was working with Menge at Heidelberg. He had an associate who did a large number of intervertebral injections into the foramen. We experimented on cadavers, and out of sixteen cases in which we attempted to inject methylene blue directly into the foramen, we were successful in four only, and in the others the injections were in the neighborhood of the foramen. Since that time, I have practiced catching the nerve just under the lower border of the rib at the posterior angle. The needle is carried forward until it comes in contact with the rib, then slipped under the lower groove of the rib, and 2 or 3 c.c. of the anesthetic are injected there. The needle is slightly withdrawn and passed to the upper portion of the rib, and another 2 or 3 c.c. of the anesthetic are injected.

I feel that local anesthesia has a widespread application in thoracic surgery, but I certainly do not think it ought to be forced in a nervous patient. I do not see why we should take any risk with general anesthesia if the vital capacity of the lung is already lowered. If there is already some interference with the air bearing capacity of a lung, I do not think that we should give any anesthetic which possibly might injure the remaining portion of that lung.

IN MEMORIAM: WILLIAM STEWART HALSTED

1852-1922

The following resolutions were adopted, May 30, 1923, on the death of Dr. William Stewart Halsted:

WHEREAS, The American Association for Thoracic Surgery has learned with profound sorrow of the death of its distinguished fellow, Dr. William Stewart Halsted, and

WHEREAS, This association desires to express suitably its intense admiration for the scholarly attainments and scientific achievements of Dr. Halsted, therefore,

Resolved, That at this, the first meeting of the association since Dr. Halsted's death, there be appointed a committee to prepare a suitable memorial setting forth the notable contributions made by him to the science and art of surgery, and

Resolved further, That this resolution and the memorial be spread on the minutes of the association and printed in the annual transactions thereof, and

Resolved further, That copies be forwarded to the members of Dr. Halsted's family, and

Resolved further, That copies of these resolutions and of the memorial be placed in the "Halsted Room" at the Johns Hopkins Hospital.

As one of the great outstanding figures of contemporary surgery in America, Dr. Halsted and his life and achievements are familiar to all students of surgery the world over. Since his death, several appreciative tributes and biographic notices have appeared in the medical and scientific journals which deal intimately with his life, his labors and personality. These publications are too recent, accessible and well known to the members of this society to make it necessary for this committee to do more than recapitulate the salient facts of his career as the grounds on which their appreciation of his merit as a great leader in surgical thought are based.

Professor Halsted was born in New York, Sept. 27, 1852, and passed away at the Johns Hopkins Hospital, the theater of his greatest professional activities, just as he was approaching his seventieth birthday. At 25, he was graduated in medicine at the College of Physicians and Surgeons (Columbia, 1877) and continued steadfast and consecrated to his profession during the following forty-five years of his life.

Dr. Halsted was of English stock, and his immediate parents had descended from a long line of cultured and gentle people who had been distinguished for generations in the social, commercial and philanthropic life of New York. From these ancestors, he had, no

doubt, inherited the character and refined tastes of the "gentleman born" which were so distinctive of his personality.

As a part of his fortunate inheritance, he also enjoyed the advantages of the best educational and cultural environments that were afforded by the schools of New York, the preparatory academy at Andover and, finally, Yale, where he was graduated in arts, in 1874.

He was given an opportunity to develop his native ability on a solid educational foundation, differing in this way from many men whose names are famous in American surgery, but who were hampered by, and suffered through, the deficiencies and defects of their early preparatory training. Dr. Halsted had not only the opportunities, but, what is better, from earliest youth he gave proof of his intelligence and ability to make good use of them. After serving an internship at the Bellevue and New York hospitals, in 1878, he undertook a course of postgraduate studies in Europe, which continued for two years, until the close of 1880. During these years, he resided in Germany, dividing his time between Vienna, Leipzig and Wurtzburg, giving his closest attention to the clinics and laboratories where Billroth, Volkmann, Thiersch, Bergmann and Miculickz were the leading masters. In Germany, he first came in contact with a powerful and thoroughly established system of educational organization in which the needs and requirements of the highest medical instruction and training were duly recognized and provided for by universities maintained and controlled by the state.

The comparison between these well conducted and thoroughly disciplined governmental institutions and the disjointed, unendowed and student supported schools, which, at that time, provided for medical education in this country, was obviously humiliating to an American. It is not surprising that it roused in Dr. Halsted a spirit of protest and a desire for reform which later became apparent in his career as teacher and investigator. He was so impressed with the efficiency of the German university organizations, with their thoroughness, discipline and methods of surgical training that he did not fail to put his experience to profit in the organization of the surgical department of the Johns Hopkins Hospital when he first took charge, in 1889.

In 1880, he returned to New York fully prepared by his two years of intensive study to engage in the practice of surgery as his definite vocation. He was young, talented and socially ditsinguished, full of energy and fired by a laudable ambition to put into practical application the large store of knowledge that he had so industriously acquired at home and abroad. His exceptional merit and ability were soon recognized, and the gates of opportunity were open wide to him in a way that seldom comes to the lot of even the most favored sons of the great metropolis. Shortly after his arrival, a series of college

and hospital appointments followed in quick succession, which gave more than ample opportunity to display his technical ability and scientific attainments. First, assistant demonstrator and then demonstrator of anatomy at the College of Physicians and Surgeons (Columbia), where he was associated in teaching with Bull, McBurney, Frank Markoe and Richard Hall, all since departed. About the same time he was appointed attending surgeon to the Charity Hospital (1881-1885) and Bellevue and Presbyterian hospitals (1885-1887); associate surgeon, Roosevelt Hospital, and surgeon in chief to the Emigrant Hospital (1881-1884). In addition to the enormous activity required to discharge the duties of these numerous and exacting positions, he organized a corps of teachers for the extramural instruction of medical students, which contributed in no small way to his prestige as a teacher with the student body. One of his distinguished pupils, the late Dr. Charles A. Powers, has left a record of the impressions that Halsted produced on the minds of the men who knew him in the eighties when he was all aglow with energy and youthful enthusiasm. In an appreciative notice, he states:

> Halsted was a remarkable teacher of anatomy. Pupils in his private class went to his office at 9 o'clock in the evening, two evenings a week, where they were "quizzed" until 11 or 12 o'clock. His patience, steadiness, diligence and, above all, his thoroughness, brought out the very best that there was in those with whom he came in contact. His thoroughness and success as a teacher are furthermore attested by the fact that, in a graduating class, ten out of the first twelve men were members of his "quiz."

In the medical journals of that period and especially in the proceedings of the New York Surgical Society, we find frequent evidences of his indefatigable activity, his initiative and original enterprise in dealing with every phase of surgery. The operative technic of the day (he describes it in his memorable Yale address [1904] on the "Training of the Surgeon") was in a state of transition, and battles between the Listerians and the anti-Listerians were still being fought. In operations in which the tourniquet was not applicable, the methods of hemostasis were primitive and elementary as compared with the present immediate control of all possible bleeding points with clamps, which he most effectively helped to introduce. Anesthesia was then restricted to the use of ether or chloroform. All technical questions and problems which were of immediate practical importance appealed to his attention, and were the subject of his critical analysis, a faculty which he possessed to a remarkable degree. In his discussion of these fundamental matters, we find evidence that foreshadows the radical improvements and innovations that were to be the fruit of his more mature labors.

It was during this early period of most intense clinical activity in New York (1880-1886), that we find his passion for experimental investigation manifesting and asserting itself, in spite of the enormous burden of the operative routine that was imposed on him by his numerous hospital and teaching appointments. He then found time to investigate and expand the surgical application of Koller's recent discovery (1884) of the anesthetic properties of cocain, and to determine, by experiments performed largely on himself, the principles which govern the action of local analgesic drugs in their application to surgical practice. These discoveries were:

1. The superiority of the intradermal as distinguished from the hypodermal method of infiltration in obtaining purely cutaneous anesthesia.

2. The value of water as a local anesthetic and the efficiency of a very dilute solution of the analgesic drug when introduced in massive doses, thus reducing the toxicity of the drug to a minimum and thereby extending the field of local anesthesia to much larger areas. In this discovery alone, he antedated Schleich by at least four years.

3. The discovery that the sectional infiltration with cocain of the trunk of a sensory or mixed nerve, in any part of its course, was equivalent to a section of the nerve, thereby suppressing sensation throughout its peripheral distribution. This was the origin of the neuro-regional method of anesthesia which is expressed today by the term "nerve blocking" and is the base on which rest all methods of regional anesthesia (including dental, paravertebral, splanchnic, epidural and spinal). It is to this method that we owe the vast and amazing expansion of the regional anesthesia at the present time.

4. The indefinite prolongation of the effect of an analgesic drug by reducing or suppressing the circulation in the infiltrated area whether by mechanical constriction or other devices. Since the discovery of epinephrin and its vasoconstrictor action, all mechanical constrictors have been discarded; the epinephrin acting as a biologic tourniquet holds the analgesic drug in the tissues and retards its absorption and diminishes its toxicity, thus giving time for the performance of painless operations of prolonged duration with all the minuteness of a perfect anatomic dissection. While the mechanical compressors are no longer necessary for this purpose, the fundamental importance of ischemia in prolonging the action of the analgesic drug remains in principle the same as when it was discovered by Halsted, in 1885.

During this early period, he also was the first to discover that the blood lost in the course of a hemorrhage could be utilized for transfusion on the same patient if the extravasated blood could be recovered and collected without contamination. It was based on this principle

of reinfusion, or "refusion" as he called it, that he was able to restore a number of patients asphyxiated with illuminating gas who were brought to him for treatment at the old Chambers Street Hospital, New York, in 1893, where he was the surgeon in charge. In these cases, his procedure was to bleed the patient copiously and after collecting the blood to reinfuse it into the patient after it had been defibrinated and filtered, and thereby sufficiently oxygenated to deprive it of its toxic properties.

In replacing the lost and purified blood to the circulation, he utilized the radial artery, thus administering an arterial centripetal infusion. The utilization of the arteries instead of the veins was suggested to Dr. Halsted by reading Hermann's "Physiology" and Hueter's "Surgery," in which arterial transfusion was advised in preference to the venous route, as a precaution against possible embolism. The results obtained by Dr. Halsted, as he reported them to the New York Surgical Society, Nov. 13, 1883, were remarkable. Patients who were comatose would immediately become conscious, and were permanently improved by the reinfusion of their own blood after it had been detoxicated by defibrination and aeration. The technic of reinfusion in hemorrhage has come back, at the present time, for various indications and is applied by a different method, but in essence it remains the same as it was first applied by Halsted more than forty-one years ago. Credit has been given to Johann Thiess of Leipzig for the method of reinfusion. This surgeon applied it to the relief of exsanguinated patients in ruptured extra-uterine pregnancy and reported his experience, in 1914, without any knowledge of Dr. Halsted's previous work which had antedated his by at least thirty-one years.

This discovery and the contributions to local and regional anesthesia only are mentioned, specifically, out of many other examples that could be quoted to illustrate the originality and searching quality of Dr. Halsted's mind as this was revealed precociously, we might say, during this early, but intensive period of his life in New York, from 1881 to 1886. This important period of his life has been, in a large measure, overshadowed by the greater achievements of his more mature career in Baltimore.

These contributions also are referred to in illustration of one of the most notable traits of Dr. Halsted's character, his modesty and his repugnance to the exploitation of his merits or to the discussion of any claims of priority, since both the contributions just mentioned, which are now recognized as of capital importance in the progress of surgery, would have remained submerged and forgotten in the great limbo of the current periodical literature, so far as he was concerned. It is only fortuitous circumstance and the delving of searchers into the history

of these notable achievements, years after they had been credited to other investigators, that revealed their real origin in Dr. Halsted.

In 1886, the career which had been so successfully and brilliantly launched in New York threatened to come to an abrupt close. An infected finger followed by neuritis of the arm, which caused him "horrible pain," led Dr. Halsted to seek relief in the analgesic effect of cocain, the value of which he had learned to appreciate by the incautious experiments on himself while investigating the surgical application of this drug. The racking pain and treacherous effects of the drug came near bringing about his physical undoing. He was thrown into a state of invalidism which obliged him to abandon all his professional work. For a while, all his ambitions and magnificent projects seemed shattered.

For more than a year, his disabilities continued, but the sun that so brightly illumined his life had been only temporarily eclipsed. The spirit of research which dominated his mind never forsook him. In 1887-1888, he found comfort and restored health in the pathologic laboratory which his eminent and appreciative friend, Dr. Welch, was directing for the future Johns Hopkins Hospital, in Baltimore. Here, under the inspiring leadership of Dr. Welch, and in company with such congenial fellow workers as Mall, Councilman, Nuttall, Walter Reed, Abbott, Flexner and many others, he found not only renewed health, but his genius for investigation received a new incentive for culture and development. "No wonder," as his eminent pupil and successor, Dr. Finney, has well stated in a recent biographic notice, "that Dr. Halsted, with his splendidly trained mind, his scientific curiosity and his wide experience and interest developed rapidly in this congenial atmosphere and was not long in embarking in his remarkable career of scientific productiveness."

In 1889, he was elected surgeon in chief of the Johns Hopkins Hospital, and his authority as head of the surgical division was further confirmed, in 1892, when, on the opening of the medical school, he was made professor of surgery.

The history of the surgical division of the Johns Hopkins Hospital, during the thirty-three years that Professor Halsted presided over its destinies, is the most eloquent tribute that can be paid to his memory. It also is the best proof of the wisdom and prophetic vision of the trustees who chose him for the task as one of that remarkable group of men, the great four, Welch, Osler, Halsted and Kelly, who, as chiefs of their respective divisions of the hospital, were entrusted at the outset with the execution and fulfilment of the founder's high ideals.

Dr. Halsted now was given an opportunity to organize and establish a school of surgery in which not only the most thorough system of

instruction was made effective for the undergraduates, but also, to a limited number of properly qualified young graduates, staff appointments were opened, which favored to the utmost the development of their technical abilities, and, at the same time, encouraged them to perfect themselves in laboratory methods of investigation and research.

The great care which was exercised in filling the vacancies on his permanent staff, by which men only of recognized ability and special aptitude for progressive surgical work were selected, is well shown by the eminence and distinction attained by the men who were privileged to be his pupils.

The thoroughness with which the selective plan of organization was carried out and the opportunities offered for pathologic, bacteriologic, physiologic and experimental research, the last in the now famous Hunterian laboratory, and the minute and exacting care given to all the details of operative technic soon yielded results which were apparent in the high order of scientific publications by the surgical staff of the hospital.

Having laid the foundation for a model training school for surgeons by the organization of its teaching and operating personnel on lines that insured its stability and efficiency, Dr. Halsted, as a next step, systematized and standardized the operative technic. Here, again, he had to deal with the fundamentals, anesthesia, hemostasis and asepsis. In each one of these, he had already laid the indelible impress of his originality. We have referred to his epochal discoveries in the domain of local and regional anesthesia. It was only quite recently, April 2, 1922, barely six months before Dr. Halsted's death, that the importance and significance of his great contribution was duly recognized by the National Dental Association, which presented him with a beautifully designed gold medal and gave him full credit as the originator of the method of neuroregional anesthesia which has proved of such incalculable service in oral and dental surgery. It is natural that under such a master of the technic of local and regional anesthesia, the Johns Hopkins Clinic should have been one of the first to utilize and develop its practical application.

While local and regional anesthesia was one of his great passions, Dr. Halsted did not overlook the importance of general anesthesia. In this also he was in the van of progress, for, Feb. 17, 1910, he participated in a symposium on thoracic surgery at the New York Academy of Medicine, in which the chief topic of discussion was the maintenance of respiration by Meltzer and Auer's method of intratracheal ventilation during intrapleural operations. In the discussion which followed the reading of important papers by Meltzer, Willy Meyer, Carrel and Elsberg, with Samuel Robinson, Henry H. Janeway, W. W. Keen,

Halsted and Bloodgood participating, Dr. Halsted supplemented his observations on the Meltzer method in the practice of thoracic surgery, by an account of his recent experience with a mixture of nitrous oxid, carbon dioxid and oxygen, which he had been using for one year in his clinics. He had used this combination in prolonging operations and had been surprised at the ease with which the patients were anesthetized under its influence and the rapidity with which they regained consciousness. Before he had used these gases, he had not realized how detrimental the use of ether was in comparison. Here we see him as a pioneer in a new field, trying and investigating a combination of anesthetic gases at a time when the gas-oxygen mixture for prolonged surgical operations was practically unknown and ether was still the universal anesthetic in surgery. This observation also shows how keen and alert his mind was to all suggestions that could improve the existing method of pain suppression in surgery.

The prevention and control of hemorrhage, the saving of blood, was another factor in the safety of surgery that gave him great concern and to which he devoted the most minute attention. In his writings, we find many passages which refer to the comparative disregard of hemorrhage and the inadequacy of the hemostatic armamentarium, as he witnessed it in his student days. He [1] states, "Rarely have I seen in our country, prior to my visit to Europe (1878), more than one artery clamp at a time, left hanging in a wound. Clamps were too few for this, four to three, or even two, being considered ample for an operation. Few hospitals in New York possessed as many as six clamps in 1880." What a contrast, with the present practice which he helped so materially to introduce, when as many as fifty and more clamps may be seen in an operative field. Halsted in his early advocacy of greater use of hemostatic clamps may be ranked again as a pioneer, reminding us in his forcible pleading of Koeberle, Pean and Spencer Wells, the fathers of the hemostatic clamp in Europe. But, while he was an energetic advocate of the hemostats, he also was discriminating in their use, as we see in another passage of his "Operative Story of Goiter," "My chief weapon against hemorrhage is the artery clamp, a finely pointed instrument which penetrates and dissects, but does not *crush* the tissues in an unnecessary manner."

This delicate, fine pointed instrument is now known as Halsted's "mosquito" forceps, which is interesting not only because of its hemostatic value but also as a reminder of the inventor's great respect for the blood and living tissues.

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1. Halsted, W. S.: Operative Story of Goiter, Reports Johns Hopkins Hosp. **19**, 1920.

His attitude toward hemorrhage and the importance of its absolute control is illustrative of his conservative surgical conscience and his constant solicitude for the patient's safety. This attitude cannot be better stated than in his own words: [2]

> One of the chief functions of surgery is the management of wounded vessels, the avoidance of hemorrhage. The only weapon with which the unconscious patient can immediately retaliate upon the incompetent surgeon is hemorrhage. If he bleeds to death, it may be presumed that the surgeon is to blame; whereas, if he dies of pneumonia, peritonitis or other infection, or from an unphysiological performance, the surgeon's incompetence may not be so evident.

We now come to the third leg of the vital tripod on which rests the whole superstructure of modern surgery, asepsis. As was to be expected from a surgeon who had contributed so materially to the advance of anesthesia and hemostasis, Professor Halsted's solicitude for the protection of his wounds from infection is apparent in all his work and writings. A convinced Listerite in his student days, when his teachers were still wrangling on the merits of the Listerian doctrines, he was one of the first surgeons of the country to grasp the full meaning of sterilization and asepsis and to adopt these with absolute conviction as articles of faith. With asepsis as a foundation, he gradually evolved a technic which, for many years, was unique in the originality of its methods, in the thoroughness and rigid discipline with which it was carried out and in the unrivaled excellence of the results which it obtained. A profound student of traumatic processes and of the reactions of wounds to infection, he was not long in devising new and ingenious methods by which he could protect his wounds and obtain the most perfect results. We need only recall that it was he who, in 1899, first introduced the use of rubber gloves in surgery, and if we consider the enormous influence that this single innovation has exercised in increasing the safety of surgery and the fact of its universal adoption by surgeons all over the civilized world, we must admit that for this service alone, Professor Halsted is entitled to a monument. It is to him also that we owe the familiar "cigaret" and "cigar" drains which have taken the place of the old rigid tubular drains, and the use of rubber tissue to protect granulations from insult; to prevent the adhesion of granulations to gauze packs; to promote healing under moist scabs, and to protect skin grafts, all of which were suggested and worked out by Halsted, as far back as 1881, when he was surgeon to the Roosevelt Hospital Dispensary, New York. The use of rubber for the protection of granulating surfaces and to prevent the adhesion of dressings involves a principle of wound treatment of no mean practical

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2. Halsted, W. S.: Effects of the Ligation of the Common Iliac Artery, Tr. Am. S. Assn., **30,** 1912.

importance which the profession as a whole has only recently begun to appreciate. The experience of the World War has more than confirmed the importance of this detail, and manufacturers, quick to seize on every improvement in technic, are now putting on the market a number of substitutes, such as paraffin sheets, celloidin tissue, silkoid and other products of greater or less value, which only confirm the value of Halsted's original idea.

Closely interwoven with Professor Halsted's methods of wound healing is the question of the material that he used for ligating blood vessels and suturing the tissues. In this regard, his preference for silk has probably excited the most discussion. With him the advantages of fine silk, threaded on fine needles, over catgut or other absorbable material were indisputable. Any one reading his argument in favor of silk versus catgut cannot fail to be impressed with the force of his reasoning, based on sound and incontravertible logic.[3] Without entering into the merits of this discussion, any one who had seen him work with his fine silk and delicate straight needles can readily understand that, in his hands, or in those of the surgeons trained by him,[4] the superiority of silk in aseptic wounds, as suture and ligation material cannot be questioned. Apart from the fine needles and quality of the silk, the reasons for his invariable success were scrupulous asepsis; the care with which the tissues were handled; the avoidance of crushing of tissue masses, and the perfect hemostasis which he secured by the transfixion of the individual vessel and its ligation with the finest of threads. It was the combination of all these factors that permitted the closure of wounds without drainage, thereby diminishing the chances of contamination. The fact that for over twenty-three years silk had been used exclusively in his clinic with practically uniform primary healing amply justified his preference for silk. To him we also owe the intracuticular suture for the cosmetic closure of wounds and his still more notable and original methods of skin grafting.

Need we quote other examples to prove Professor Halsted's inventiveness and his influence in remodeling and recasting the older technic of surgery? It has been said that Dr. Halsted leaned more to the science than to the art of surgery and this, no doubt, is true so far as his natural inclinations were concerned, but it is difficult to decide which of the two he benefited most. With the proof before us of what he did to improve the methods that are fundamental and common to all modern operations, how can we question his profound interest in

3. Halsted, W. S.: Ligature and Suture Material; the Employment of Fine Silk in Preference to Catgut, J. A. M. A. **60**:1119-1126 (April 12) 1913.

4. Taylor, A. S.: The Use of Fine Silk in Surgery, China M. J. **35**:5 (Sept.) 1921.

the art of surgery with the evidence that he has given us of his perpetual concern, almost to the hour of his death, in perfecting more complex major operative procedures? Need we recall what he did when, a little over a quarter of a century ago, he revolutionized the surgical treatment of cancer of the breast?

The same interest, originality and practical value is displayed in his operations for the radical cure of hernia, for the resection and suture of the intestines, for the drainage and suture of the common bile duct, in which, as in most of his operations, he thought and worked long in advance of his time. In his operations on the thyroid for the cure of goiter, and his ligation of the inferior thyroid arteries (which few surgeons have adopted in spite of its physiologic superiority, because the upper bipolar ligations as so much easier), in his success in the occlusion by aluminum bands of the aorta and other dangerous arteries, we find enduring testimonials of his love of the art as well as of the science of his profession.

Looked at from the point of view of the art, Halsted as an operator has well been described as the father of the school of the surgery of safety, a school which insisted on a technic that subordinated everything to the safety and welfare of the patient. It put in force the most rigorous asepsis and the most uncompromising discipline in guarding the tissues from insult, by scrupulous attention to all details, however, small, that might affect the tissue or the thoroughness and finish with which the operative act was carried out.

Leriche, in his tribute to Halsted, says:

> "Beginning at a time when surgery was still living under the spell of the preanesthetic days, when rapidity of execution was awarded the highest premium, he stood firm on the ground that the nature of the material upon which the surgeon exercises his handicraft is too noble and too precious to be insulted with impunity or to allow even the smallest living particle to be wasted; and to do this effectively, the surgeon must subordinate brilliancy and rapidity to thoroughness and safety. . . As an investigator, Halsted understood surgery as experimental science, aiming at, as his objective, the greater and deeper knowledge of the laws that govern physiological and pathological life. Halsted saw pathological and clinical problems everywhere that invited investigation and that he made the object of experimental researches which he carried out with patience, painstaking labor and with all the discipline of a Claude Bernard. After settling on his problem, he thought it out to himself. He looked at it from every angle, but first objectively as physiologist and then as a clinician."

The qualities of thoroughness, absolute scientific honesty, accuracy and vision which characterized Dr. Halsted in all his undertakings gave to his conclusions the greatest value. In this respect, his investigating and inquiring turn of mind reminded one of John Hunter, whom he admired above all the surgeons of the past. Not unlike Hunter, he

could well say, when facing an obscure problem which could not be solved by mere theorizing, "Why think, why not try the experiment?"

Dr. Halsted's literary productions were characterized by the same distinctive, painstaking thoroughness and attention to detail which was peculiar to all his work. His major monographs, "On the Surgical Treatment of Cancer of the Breast," "On the Surgical Treatment of Goiter," "On the Surgery of the Left Subclavian Artery," "On the Surgery of the Common Iliac," and "On the Partial Occlusion of the Aorta and Other Great Arteries by Aluminum Bands," are universally recognized as epochal in importance and value, not only in virtue of his own original contributions to the subject, but because they embodied all the records of previous operations performed by other operators for the same conditions. In this way, his complete monographs represent an immense amount of bibliographic research being, not mere compilations, but the most critical analyses with commentaries and criticisms on each case, which add immensely to the value of the compilation. His comments and crictisms, usually short and pithy, are most illuminating and are the very kernel of the knowledge one is seeking. The great labor and time required in the preparations of these opera magna necessarily limited his literary output to a relatively small number of yearly productions, but each one of these works is a landmark in the history of the subject. He was well aware that the surgical world had learned to expect great things of him, and he spared no labor even to exceed the level of the highest expectation.

Modest, self repressed, shunning the limelight of publicity, he never obtruded his personality or exploited his deeds and only referred to himself in the most impersonal way, allowing the facts always to speak for themselves. In this, he seemed to have inherited the Baconian precept that "a man can scarce allege his own merits with modesty, much less extol them. Such things are grateful in a friend's mouth which are blushing in a man's own." Though shy and reserved and undemonstrative, caring little for the "gregarious gatherings of men," as Cushing expresses it, and living largely to himself, his home, his laboratory and his clinic, he delighted in the company of his pupils, immediate associates and a few chosen friends. With these, he was expansive, and the glow and warmth of his friendship rapidly dissipated whatever restraint might first have been imposed by his punctilious politeness and adhesion to conventional formalities. To those who were privileged to bask in the sunshine of his friendship, the true nature of the man was revealed in all its splendor. When he allowed his mind to expand freely in unrestrained confidence, his breadth of thought, his wide culture, his intimate knowledge of the greatest leaders of the profession throughout the world, whom he met in his frequent travels, added to the amenity and suggestiveness of his conversation. He was

generous, liberal and hospitable to a fault, and his kindliness, sympathy and unequaled liberality in helping the younger men to accomplish great undertakings by encouraging them through privileges and opportunities which he secured for them are all qualities which account in a great measure for the admiration and loyalty that is so notably displayed by his associates and pupils.

Dr. Halsted has given proof of his genius in many ways. He was great in his art. He was great in his science. He was great in his rare appreciation of the unity of the art and science of surgery and of the correlation of the medical sciences in general. This gave him an unique distinction as a teacher of surgery. He was great as a father and founder of a school of surgery which has ever stood foremost in surgical scholarship, in surgical craft and in the attainment of surgical ideals and achievements. In none of these, however, was he greater than in the wisdom he displayed in selecting the group of young men whom he chose to be his apostles, and to transmit his teachings.

"By their fruits shall ye know them," says the Evangel. By the fruits of his labors, by his written contributions, by his indelible impress on the surgery of today and of all time, we, the American Association for Thoracic Surgery, know that William Stewart Halsted, though departed, yet abides, an immortal in his chosen realm of surgery.

GEORGE JULIUS HEUER, M.D.
ROBERT TALBOTT MILLER, M.D.
RUDOLPH MATAS, M.D. (Chairman)

TREATMENT OF PULMONARY TUBERCULOSIS WITH THE HELP OF ARTIFICIAL PNEUMOTHORAX

CONSIDERATION OF END-RESULTS

SIDNEY F. BLANCHET, M.D.

SARANAC LAKE, N. Y.

The treatment of pulmonary tuberculosis by compression of the diseased lung through artificial pneumothorax has been on trial for over thirty years. Those who first took up this method did not do or report much before the year 1900. Nevertheless, their results keenly interested a number of others, and, from 1900 to 1910, artificial pneumothorax was applied in many cases of pulmonary tuberculosis in Europe and in not a few in America. During the following decade, from 1910 to 1920, pretty generally, workers in tuberculosis consistently employed this mode of treatment.

In judging the permanence of results following the employment of artificial pneumothorax treatment, it is only fair (as in judging results following the usual treatment) to wait, after the completion of the treatment, until a sufficient time has elapsed to prove that the results are permanent.

My own experience with artificial pneumothorax began in 1911. The work has all been done in private practice. Two hundred cases are reviewed in this report, and, in all, the treatment was completed at varying periods between 1912 and 1922. An approximate idea of the percentage of cases suitable for artificial pneumothorax can be had in my experience from the fact that out of 2,000 cases of tuberculosis in all stages treated during those ten years, in 200, that is, in 10 per cent., it seemed worthy of trial. This percentage is rather deceiving because this measure was probably indicated and would have been successful in many more of these 2,000 cases, if the patients had come for treatment before their disease became far advanced.

Each time I have attempted to draw up figures that would give a fair idea of the end-results to be expected from the use of artificial pneumothorax, I have been confronted by the fact that if all cases treated are reported in one group, these figures are not just to artificial pneumothorax. When this measure is applied in far advanced cases, even if a good compression is secured, the results are very often disappointing because the disease has gone too far to respond to any treatment. In these advanced cases, if adhesions do not interfere with the development of an effective pneumothorax splint, we often have to contend with serious complications in other parts of the body, such as tuberculosis of the larynx or the intestines, which may have developed

seriously enough to prevent the patient's responding to the splitting of the diseased lung. So that the results from artificial pneumothorax when it is applied in this far advanced group cannot fairly be given together with the results obtained *when it is given in time,* that is, in cases which are moderately advanced. Therefore, I have carefully separated these two groups, and in the results reported here have tabulated separately the results in moderately advanced and the results in far advanced cases.

While many advanced cases must be considered, the lack of results secured in this group will make one wary in his selection of such cases. It is especially in this group that failure results on account of extensive adhesions. It is also in this group that the large percentage of serious complications, such as perforation or rupture of the lung, purulent effusion or fatal hemoptysis, occurs. Some of these complications add tremendously to the suffering and discomfort of the patient, so that, to use the words of the Scripture, "the last condition of this man is worse than the first." Although a fair percentage of patients may show some improvement, this improvement is usually more or less temporary. On the other hand, when a fatal outcome is the only thing ahead of these patients, one is often driven to make the attempt. It is at least daring some treatment, and, when a patient is very weak, a trial of artificial pneumothorax is safer than thoracoplasty. If, by this means, temporary improvement can be secured, if the patient gathers a little strength, then thoracoplasty can be more safely attempted.

My 200 cases fall easily into two groups: Group 1 is made up of 100 cases of moderately advanced pulmonary tuberculosis and Group 2 of 100 cases of advanced pulmonary tuberculosis. As one would expect, the disease in the good lung was slight or inactive in Group 1, but in Group 2 it was often fairly marked and sometimes active. While about half these patients have too recently discontinued treatment for a fair opinion to be given as to ultimate results, in at least half of them treatment has been discontinued for five years, so that in reporting the whole group, the figures ought to be of some value.

Briefly stated, of the 100 cases of advanced pulmonary tuberculosis treated during the ten years ending in 1922, ninety-seven are dead and three living. Of the ninety-seven that died, really good results followed for a time in only twelve cases, but even in these the relief was temporary, from three months in some to one year in others. However, it is fair to say that in 15 per cent. some benefit was received, while in the remaining 85 per cent. little or no improvement followed. It is well to emphasize again at this point the hopeless outlook of all of these advanced cases; in a ten year period without artificial pneumothorax, in the ordinary course of events most of them would be dead anyway.

In my 100 cases of moderately advanced pulmonary tuberculosis, the statistics of results are entirely different. Out of this 100, twenty-four patients are dead; six are failing or stationary; twenty-three, decidedly improved; in eighteen the disease is arrested, and twenty-nine are working. That is, while 30 per cent. are dead or failing, 70 per cent. have shown either decided improvement, arrest or ability to work.

These results are given in Table 1.

It seems wise to me to give these general figures on end-results with artificial pneumothorax before discussing some end-results in detail, and to describe some reasons for failure and some reasons for success in this group of 200 cases.

Taking, first, the far advanced group, in the ninety-seven patients who are dead, the most common cause of failure, as far as artificial

TABLE 1.—*Results in Two Hundred Cases of Artificial Pneumothorax Treatment, Treated During the Years 1911 to 1922*

	In One Hundred Moderately Advanced Cases	In One Hundred Advanced Cases
Dead	24	97
Failing	6	1
Improved	23	1
Arrested	18	1
Working	29	0
	100	100

pneumothorax is concerned, was the presence of adhesions that prevented an effective compression. In forty cases, the compression secured was 25 per cent. or less, and, in most of these, despite this partial compression, the disease progressed steadily to a fatal end. In twenty of the fatal cases, death followed a rupture of the compressed lung. In another eighteen cases, the patient failed more rapidly because of the development of tuberculous purulent effusion. In nine cases, a progressive development of tuberculosis in the good lung led to death. In six, death was due to hemoptysis which was uncontrolled by the compression. And, finally, in the remaining four fatal cases, death was due to influenza.

If these figures are analyzed, it will be seen that failure is most often due, first, to extensive adhesions preventing a successful application of the treatment, and, secondly, to the common occurrence in the far advanced group of serious complications. Nevertheless, as before stated, artificial pneumothorax must often be dared in this group, and even if experience gives little promise of success in the large

majority, these patients are usually grateful that some treatment is being attempted. If they are warned of the uncertainty of results, they will bear no ill will because of the unfavorable outcome.

If the failures in the group of 100 more favorable cases are now studied, of the thirty patients that are dead or failing, failure was due to an ineffectual compression in fourteen cases, to rupture of the lung in four, to tuberculous purulent effusion in seven, to hemoptysis in two, to influenza in two and, finally, to development of disease in the better lung in one case.

An analysis of these cases of failure in the more favorable group shows that lack of success in securing an effective compression was the reason in only fourteen, compared to forty cases in the advanced group; rupture of the lung occurred in four cases, while among the advanced it appeared twenty times, and tuberculous purulent effusion appeared in

TABLE 2.—*Causes of Failure of Artificial Pneumothorax Treatment*

	In Moderately Advanced Group (24 Deaths 6 Failing)	In Advanced Group (97 Deaths)
Ineffectual compression	14	40
Rupture of lung	4	20
Purulent effusion	7	18
Hemoptysis	2	6
Intercurrent disease	2	4
Development of disease in good lung	1	9
	30	97

seven cases of this group, compared to eighteen of the other (this is not including the cases of ruptured lung). Comparing those cases that failed directly as a result of progressive development of disease in the good lung, there was one case in the favorable group to nine in the advanced. Therefore, in my experience, failure was rarely directly due to the development of disease in the good lung; quite often the signs of disease in the good lung improved or cleared up. The causes of failure are given in Table 2.

Before this subject is dismissed, one cause of failure which is difficult to estimate should be at least mentioned. Very often, the patient who responds unusually well to artificial pneumothorax is too much reassured by his improvement; he feels so well that he becomes overconfident and careless. As a result, this patient does not carry out the rest treatment in the faithful way that is necessary to give him the best chance to build up and so get the maximum amount of repair or healing in the diseased lung during the time it is splinted by artificial pneumothorax. This is so

often the case that justice in reporting the results of this treatment cannot be fully given without separately tabulating carelessness on the part of the patient as a possible cause of failure.

In only one of the 200 cases reported was death due to pleural shock. This case was one of far advanced tuberculosis, very unfavorable for treatment. The accident happened, at the time of the ninth injection, when the needle entered the pleura and before either manometer or gas had been turned on. Procain had been employed apparently with the usual success.

In one case, there were symptoms that suggested air embolus, but these soon passed off and left no after-effects.

In two cases, the patient fainted after the introduction of the needle. But both of these patients were sitting up for the operation and had eaten a meal shortly before.

Among my cases of purulent effusion, examination of the fluid showed it to be either sterile or to contain tubercle bacilli. Mixed infection never occurred except in cases in which the lung perforated or ruptured. In my opinion, with a careful technic and good fortune in avoiding intercurrent infections, this calamity can be avoided.

It would be unjust not to mention the valuable aid received in a number of cases from the use of the mercury quartz lamp. Real improvement, in some cases, did not begin to appear until heliotherapy by this means was added to the artificial pneumothorax treatment. In at least twelve cases in which intestinal tuberculosis was a complication, improvement would not have followed if this complication had not been controlled by artificial sunlight treatment.

CONCLUSIONS

In conclusion, I should like to state that:

1. My figures and experience convince me that although artificial pneumothorax must be used in advanced tuberculosis, lasting results rarely follow its employment at this stage.

2. If it is used in moderately advanced cases, which fail to respond reasonably soon to the usual treatment or where, in these cases, cavities are beginning to form, a high percentage of success can be confidently looked for if adhesions do not interfere with the development of an effective compression. By arresting a well developed or progressive disease, by controlling hemoptysis or preventing its reoccurrence, a quicker, safer recovery is ensured. Also, it is well to emphasize that careful following of the usual sanatorium treatment is absolutely necessary; it adds greatly to a patient's chance of getting the most benefit from artificial pneumothorax treatment.

3. As time has gone by, I have come to believe that there is an optimal length of time during which this compression should be kept

up. If, after perhaps two or three years, one is doubtful as to the safety of allowing the lung to reexpand and function again, a thoracoplastic operation, which will make the compression permanent, is definitely indicated. This is particularly true in the case of patients whose general condition is none too good, either because they have had to work or because they have been careless and gone beyond their strength. This, in my experience, is not rarely the case. The danger of the compression longer continued is the development of purulent effusion or perforation of the lung. I have particularly in mind the twelve advanced patients that showed good improvement for a time, and also most of the thirty patients in the moderately advanced group who failed or died. A fair proportion of these forty-two patients would undoubtedly be alive today if thoracoplasty had been performed.

Finally, it is difficult to overestimate the importance of the place that artificial pneumothorax has won for itself. Already, many valuable lives have been lengthened or saved by this means. In a disease like pulmonary tuberculosis in which progress toward recovery is so long and slow, so beset with pitfalls, the development of a measure of such value has indeed been a boon to those of us whose work it is to treat this disease.

PNEUMOTHORAX *

J. J. SINGER, M.D.
ST. LOUIS

Rivier [1] states, "No more hopeful ray of sunshine has ever come to illumine the dark kingdom of disease than that introduced into the path of the consumptive through the discovery of artificial pneumothorax."

When we consider the hopeless outlook of thousands of tuberculous patients, the results of pneumothorax treatment are almost miraculous. Fortunately, the poor results formerly obtained are due to preventable causes and errors in judgment, and with increased experience even greater benefits will follow.

Originally, collapse therapy was confined to the treatment of advanced pulmonary tuberculosis when all other means had failed, but gradually it was recognized that it could be used in more favorable cases, and today all recognized phthisiologists obtain results that were formerly undreamed of. Benefits obtained are in direct proportion to the care exercised in choosing suitable cases and the judgment of the operator in his method of procedure.

It is not the purpose of this paper to go into great detail concerning the operation itself, but only to mention a few of the important facts concerning pneumothorax in general.

The cases of tuberculosis suitable for treatment fall into three classes: (1) hemorrhage cases, when there is no doubt from which lung the bleeding occurs; (2) unilateral cases with a good compensatory contralateral lung, and (3) bilateral cases in which cough and serious symptoms of toxemia might be relieved by the partial collapse of one lung, or even of both lungs.

In Class 1, that is the hemorrhage cases, brilliant results are obtained. Frequently, aspiration pneumonia and rapid spreading of the tuberculous process follows the hemorrhage. One must use more air in these cases because it is necessary to obtain a complete collapse as soon as possible. The usual method of combating hemorrhage by using hemostatic drugs and even transfusion frequently fails when a single pneumothorax treatment produces the desired result. The great stumbling block to satisfactory collapse is adhesions between the pleurae. One is never sure of the presence or absence of adhesions before the use of the manometer.

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* From the departments of internal medicine and surgery, Washington University Medical School, St. Louis.

1. Rivier: Pneumothorax Treatment.

However, if no free pleural space is found in the first attempt, several other sites should be tried before discontinuing treatment.

In Class 2, the unilateral cases, a definite localization of the tuberculous process in the one lung is rare, but experience has shown that, in the presence of a small amount of infection in the contralateral lung, good results will frequently follow the treatment. It is understood, of course, even in this class of cases, that the usual expectant rest treatment has been carried out with no benefit. One should not wait too long, however, as it is in precisely these cases that good judgment is so necessary to obtain good results.

In Class 3, the far advanced bilateral cases, the most hopeless sufferers are often placed in the position of enjoying, in a way, the few remaining months or years. Many an unfortunate patient is allowed to suffer on and on, especially with the persistent cough and sweats, when he might be much improved by judicial use of pneumothorax treatment. It has been shown, recently, by Stivelman [2] and others, that partial collapse will do much more than was expected. Observation of this class of cases with partial collapse is that the air introduced produces most compression on the diseased area and less on the healthy part of the lung. This seemingly selective collapse is explained on the theory that diseased tissue is less elastic than the healthy parts of the lung and, consequently, offers less resistance to pressure of the gas. These conditions in the chest can be readily noted in the fluoroscopic examination.

Statistics show that this class of cases gives the largest percentage of failures as far as cure is concerned; yet, at times, startlingly good results are seen even here.

One frequently hears that the benefits derived are due to the psychologic effects of "doing something." One cannot deny that a certain percentage of persons react in this way, but the results are usually due to actual compression of the lung itself.

Another important use for pneumothorax is for diagnostic purposes. It has been noted, by many workers, that more information of the lung pathologic condition can be had by the introduction of a small amount of air in the pleural cavity in obscure cases when fluid is present. By removal of the fluid and the replacing of it with air, tumors of the chest wall and of the lung, mediastinal masses and calcification of the pleura can frequently be seen. In this method, it is not necessary to introduce a large amount of air, and it can very readily be removed after the desired information is obtained.

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2. Stivelman, B. P., and Hennel, H.: Am. Rev. Tuberc. **7**:290-306 (July) 1923.

It has been observed frequently that following repeated aspiration of fluid and frequent recurrence, the introduction of from 200 to 500 c.c. of air will prevent further accumulation of fluid, and, as the air is absorbed, the lung will reexpand. It also has been noted that where aspiration of fluid from the chest cavity is difficult, the introduction of air causes a back pressure that will make aspiration easier.

COMPLICATIONS

It must be emphatically stated that pneumothorax is reserved for cases that have not responded to the ordinary treatment. When an operation is done there are certain complications that must be expected from time to time. The most important complications are serofibrinous pleurisy, interstitial emphysema, tuberculous empyema, nontuberculous empyema, adhesions preventing collapse, spontaneous pneumothorax, air embolism, hemorrhages and death. Fortunately, serious complications are relatively rare; most of them are of no great importance. As will be seen by referring to the tables, serofibrinous pleurisy is the most common; only occasionally will the fluid be sufficient in amount to require aspiration. Occasionally, interstitial emphysema will occur because of the tearing of the adhesions. Through violent cough and the tearing of the lung tissue, the infection takes place through the bronchial tubes. The most serious complication is embolism; this may produce sudden death. Several explanations have been given for this unfortunate occurrence, but none seem to be perfectly satisfactory.

Schlaepfer,[3] in a recent article, states that air is introduced into the systematic circulation if the needle is introduced into vessels of adhesions and carried to the cerebral vessels. By careful observation of the manometer during the introduction of air, one can be certain that the needle is in the pleural cavity, and thus avoid trouble. Hemorrhage of the opposite lung occasionally occurs. This, however, is unavoidable as far as the operator is concerned. The explanation for this complication is that the contralateral lung takes on hyperactivity and latent ulcerated parts of the lung may be torn by excessive coughing. When death occurs, it is usually due to embolism.

RESULTS

Tabulated results definitely show that pneumothorax, properly used, will save many lives and give great comfort to many tuberculous patients.

The tables were compiled from five charts giving the results of several workers in this field. The analysis of Chart 1 showed definitely

3. Schlaepfer, K.: Bull. Johns Hopkins Hosp. **33**:321 (Sept.) 1922.

that cases with minimal involvement offer the best chance for apparent cure (Table 1). A large number of moderately advanced cases in

TABLE 1.—*Improvement After Pneumothorax in Various Stages of Tuberculosis (Author's Cases)*

	Stages	Per Cent.
Incipient	5	14+
Moderately advanced	12	34+
Advanced	18	51+
	Incipient Cases	**Per Cent.**
Apparently well	4	80
Not improved	1	20
Deaths	0	
	Moderately Advanced	**Per Cent.**
Apparently well	7	58+
Improved	3	25
Not improved	2	16+
Deaths	0	
	Advanced	**Per Cent.**
Apparently well	3	16+
Not improved	4	22
Deaths	11	61

which pneumothorax is tried show a small percentage of the cases improved. Results obtained bear out the statement that, while advanced cases more or less suitable for treatment are found, the earlier in the progress of the disease that pneumothorax is used the greater the improvement to be expected. Incipient cases, with reference, not to time, but to the amount of involvement, give the best results. Deaths noted in the chart are not deaths due to operation but to the tuberculous process.

Table 2 gives the various complications arising in this series of cases.

TABLE 2.—*Complications*

Serofibrinous pleurisy	11
Interstitial emphysema	1
Empyema, tuberculous	1
Empyema, nontuberculous	1
Adhesions preventing collapse	7
Spontaneous pneumothorax	2
Air embolism	2
Hemorrhages	1
Deaths	1

In Table 3, one can very definitely see that, in patients in whom pneumothorax was attempted but was found not possible on account

of adhesions, there were 88.4 per cent. deaths in comparison to 48.4 per cent. in whom pneumothorax was possible.

TABLE 3.—*Results* (Foster, Webb and Gilbert[4])*

1. Unsatisfactory collapse	36	cases
Living, but not well	2	
Well, not working	2	
Dead	32	
2. Reasonable or complete collapse	62	cases
Dead from progressive tuberculosis	25	
Dead from complications.		
Absorption	1	
Syphilis	1	
Embolism	1	
Tuberculous enteritis	1	
Thoracoplasty	1	
Alive, but not well	13	
Living and working	19	

*Of these, sixteen have stopped treatment; three continue treatment. Treatments began in August, 1912; January, 1913, and July, 1914.

Of those who had fair to excellent collapse, sixty-two, the results in percentages are:

		Per Cent.
Well and at work	19	30.6
Partial result, improved	13	21.0
Dead	30	48.4

Of those in whom there was no free pleural space, or one so slight as to preclude treatment, thirty-six:

Well and able to work	2	5.5
Alive, but not able to work	2	5.5
Dead	32	88.8

Table 4 gives another series of cases showing 70 per cent. improved or well from pneumothorax.

TABLE 4.—*Another Series of Results (Cunningham, Ware and Smith[5])*

Thirty-three cases reported:		Per Cent.
Dead	2	6.66
Lost	2	6.66
Living:		
Unimproved	6	18.18
Improved	11	33.33
Well	12	36.36
Fluid	22	66.66

Table 5 is self explanatory, and there is no question from these statistics that great benefits result in cases of tuberculosis either from partial or complete pneumothorax.

4. Foster, Webb and Gilbert: Tr. Nat. Tuberc. Assn., June, 1923, p. 95.
5. Cunningham, Ware and Smith: Tr. Nat. Tuberc. Assn., January, 1923.

TABLE 5.—*Results Showing Benefits from Partial or Complete Pneumothorax (Matson, Matson and Bisailon[6])*

Six hundred cases:
149 are clinically well
84 are arrested ambulant
274 are dead

The remaining cases are either favorably influenced, temporarily improved or unimproved.

Of the one hundred and forty-nine cases clinically well:
114 were satisfactory compressions
28 were partial compressions
7 had no free pleural space

Of the eighty-four cases arrested ambulant:
44 were satisfactory compressions
29 were partial compressions
11 had no free pleural space

Of the two hundred and seventy-four fatal cases:
52 were satisfactory compressions
142 were partial compressions
80 had no free pleural space

Of the two hundred and ten satisfactory compressions:
114 are clinically well
44 are arrested; ambulant
52 are dead

Of the one hundred and ninety-nine partial compressions:
28 are clinically well
29 are arrested; ambulant
142 are dead

Of the ninety-eight cases no free pleural space (control cases):
7 are clinically well
11 are arrested; ambulant
80 are dead

CONCLUSIONS

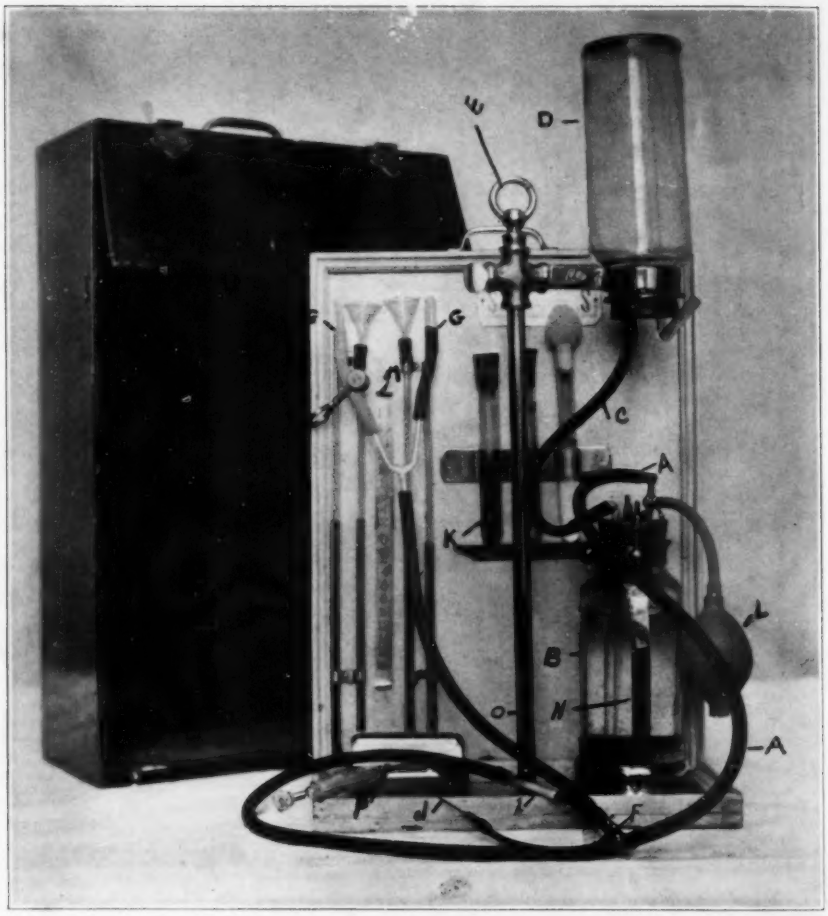
1. All evidence points to the great value of pneumothorax in the treatment of pulmonary tuberculosis.

2. Complications arising are relatively rare in the hands of careful operators.

3. Diagnostic pneumothorax is valuable in pulmonary involvement.

4. Pneumothorax should be instituted early in cases showing no improvement after at least six months of the usual treatment for tuberculosis.

6. Matson, Matson and Bisailon: Tr. Nat. Tuberc. Assn., June, 1923, p. 118.



A, outlet from lower bottle *B*; *C*, connection of upper bottle, *D*. This is adjusted in height by a screw at connection, *E*. For the enclosing case, this bottle is lowered so that it fits in the case. *F*, a two-way valve which permits the air to flow from *B*, and also connects with manometers, *G*; *I*, a piece of glass tubing with sterile cotton to filter the air before passing through the needle, *J*. *K*, tube stand fastened to the back of the instrument; this has three clips which hold three test tubes, containing iodin, alcohol and sterile procain. *L*, bulb connected with the two-way valve through which air is pumped into the lower bottle, thus forcing the water into the upper bottle. When all the water from the upper bottle has dropped into the lower bottle, the fluid is again pumped by the use of *L* into the upper bottle. It can be seen that, by this method, a large bottle is not necessary. *M*, two small funnels through which fluid is introduced into the manometers; *N*, metal scale attached to the base holding the lower bottle; *O*, stand that supports the upper bottle in various positions, and *P*, box containing the hypodermic syringe for local anesthesia.

PORTABLE PNEUMOTHORAX APPARATUS

The important features of the Singer portable pneumothorax apparatus are:

1. There are two manometer tubes, 30 inches (76.2 cm.) in length, allowing for greater pressure.

2. There is a sliding scale between the two manometer tubes serving either side. The scale is marked, corrected for immediate calculation.

3. The two-way valve bulb attached to the upright bottle obviates the necessity of having large bottles, thus decreasing the weight of the apparatus.

4. The scale attached to the upright stand is an integral part of the base.

5. In case of breaking a bottle, any 500 c.c. druggist's display bottle can be used and refitted in the base.

6. The rubber cork is fastened to the bottle with clamps so that no leakage may occur.

7. The rack for test tubes carries all solutions necessary in the operation.

8. An attachment for the hypodermic case filled with a syringe and necessary needles is a part of the apparatus.

9. The safety valve in the tube to the manometer prevents the manometer fluid from escaping when the patient coughs.

10. The extra manometer is always in readiness; this is a very definite advantage.

11. The entire apparatus on the stand represents the back of the case.

12. The entire apparatus weighs only ten pounds; there are no expensive parts to renew.

The instrument is made by William A. Philips Company, St. Louis.

Metropolitan Building.

DISCUSSION ON PAPERS OF DRS. BLANCHET AND SINGER

Dr. Willis S. Lemon, Rochester, Minn.: The treatment by pneumothorax is, of course, simply that of rest, of a modified type. Rest has been considered a primary requisite in the treatment of tuberculosis for a great many years and has been modified by many mechanical devices; for instance, sand bags, jackets of various type, bandages, as directed by Dr. Sewell, and plaster jackets, as designed by Dr. Kinsella and shown by Dr. Forester of Colorado Springs. We must consider, then, that pneumothorax belongs to this group of mechanical devices to place the lung at rest. Such methods are applied to a rather small group of cases, since they do not affect more than from 5 to 10 per cent. of the patients. In considering the end-results of treatment, Miner estimates that about 12.5 per cent. are cured; Ringer, 13 per cent., and various authors give the usual average high percentage of about 20. Thus, about 1 per cent. of the vast number of persons suffering from moderately advanced or advanced tuberculosis are cured by pneumothorax.

The next step in the rest treatment of the lung has to be given over to the surgeon, and consists of thoracoplasty, which is strongly advocated by Sauerbruch, who has treated a large number of patients in this manner. These patients probably represent 1 or 2 per cent. of the entire number treated.

Before considering the end-results, one should consider what is actually happening in a lung that has been compressed. As an immediate consequence, I believe symptoms are likely to be increased rather than decreased, because in the collapse there is a good deal of toxic material squeezed out through the lymph and into the circulation. Symptoms ordinarily mean that there is an exchange of tissue fluids between the lesion itself and the systemic circulation. However, following the time of the initial decrease of symptoms, there is a spectacular and rather marvelous good effect of the lessening of the interchange, and the marking down, as it were, of the amount of material that is absorbed in the body. And herein lies a certain danger to the unwary, because the patient feels extremely well, and the physician is inclined to think of the symptoms rather than the lesion.

Any one but an expert, or any one who is not constantly thinking of what is actually inside the chest, would do well to visit a pathologic laboratory and actually see the lungs that have been treated — the fibrocaseous condition of that lung; the nodular condition that spreads throughout the lung; the evidence of rarefaction; the formation of cavities, many times even of gelatinous pneumonia, and the area surrounding the lesion where the tissue is least resistant — and then determine the advice to be given the patient, knowing that these end-results may be approached even with the lung in collapse. After all, these symptoms of well-being that seem so spectacular cannot mean a cure of the disease, but only that the tissue fluid exchange is lessened.

There are certain dangers associated with this work, not in the hands of the experts we have been listening to, but in the hands of a large number of physicians who deal with tuberculous patients, outside of institutions. Lack of experience and skill is one danger. The effect on the discipline of the patient himself is another danger. As has been implied, the patient, feeling well, sees no reason why he should not go back to work. Colonel Bruns tells me that the most serious problem he has to contend with is that patients do not wish to be further hospitalized. I am glad to have Dr. Blanchet say that he feels these patients should be treated in sanitoriums, that they should be cared for as if they did not have pneumothorax, and that the treatment should be continued for as long as is usually necessary to cure such a pathologic lesion. This one precaution particularly should be employed. Pneumothorax should be given because it gives immediate, decided benefit, and because in many cases it not only contributes to well-being, but also actually cures. Finally, it should be given preferably in an institution because one is treating a lesion that takes a long time to get well, and close observation is obligatory.

Dr. Everett K. Geer, St. Paul: The two high points that I have seen in these papers have been, first, that our best results are obtained in the moderately advanced case, which means that the earlier we collapse our tuberculous lungs the better results we will get. As soon as we can identify a tuberculosis lesion as being a progressive one and by a progressive lesion I mean one which shows no improvement with absolute rest in bed in the open air, it seems to me that pneumothorax should be considered and instituted. The second point is that these patients should be sanatoriumized; by far the best results are obtained in institutions.

I am sorry that the problem of pleural effusion was not dwelt on longer. That is a question that I am still very much up in the air about. I have seen pleural effusions do some very peculiar things, and I have been at an utter loss to explain just why certain things happen. I have tried going at these fluids in different ways. When some patients began effusing, I would let them entirely alone, permitting the pneumothorax cavity to fill completely. One of these, after she had stayed filled for a year, within two weeks absorbed all her fluid, and the lung came out. Another filled up for a year, and then her lung began to reexpand; I aspirated the whole thing, replaced with air, and she has not effused since. Others, I have attempted to aspirate when they have filled up, say to the fourth rib; some of them will keep on effusing, and some of them will stop. I was interested in Dr. Bettman's paper on this question, because I hope that eventually he will be able to help us solve this perplexing question.

The problem of purulent effusions is one that I would like to have the surgeons say something about, particularly Dr. Hedblom. I have done everything that I could find suggested in the literature, and have probably been more successful with formaldehyd and glycerin than with anything else. In two patients, I have stopped effusion completely with formaldehyd and glycerin, by repeated injections and aspirations.

Dr. David A. Stewart, Ninette, Manit.: My experience in artificial pneumothorax runs back over ten years and over about 260 cases; at any given time in the last few years, I have had under treatment from forty to sixty patients.

Even the men interested in chest surgery might learn chest pathology by seeing numbers of pneumothorax cases at different stages in the fluoroscope. The use of the fluoroscope before and after fills and refills is a very excellent means of controlling these cases.

As to percentage, Dr. Blanchet spoke of 10 per cent., Dr. Singer of 5. It all depends on the kind of cases you are dealing with. If you have a fairly large percentage of advanced cases, you should apply pneumothorax in even a greater percentage of cases than 10. Out of 270 patients with about 185 in bed, I find that our percentage of pneumothorax cases runs over 20 per cent.

Of course, the number of patients cured by pneumothorax is comparatively small because the possibility of real cure in such advanced cases is small; but I think that the more one uses the method, the more one is inclined to say that the benefit through pneumothorax approximates 50 per cent. That is, whatever your patients would have done otherwise, patients suitable for pneumothorax have their chances of recovery improved 50 per cent. by the use of that method. This estimate is low.

Perseverance will be rewarded in these cases. When one has failed once or twice, it is worth trying again; and when a pneumothorax has been given up, and one thinks it is impossible to renew it, it is sometimes possible to get a good pneumothorax a second time.

Adhesions are troublesome and limit the effect of the treatment in many cases, but patients and a fair amount of pressure frequently renewed will practically obliterate the smaller adhesions. They become more and more attenuated and negligible or finally snap without any harm being done. The larger ones, of course, are troublesome but even these sometimes give way to pressure. Small adhesions do not need the method of Jacobaeus; larger ones often do not, and the adhesions that do not in time attenuate are too large to be dealt with by Jacobaeus' method anyway.

In the question of which side to collapse in case of hemorrhage one is sometimes in doubt. Of course if one knows which side the hemorrhage has come from, the indication is clear; but if the side from which the hemorrhage has come should be in doubt, one usually can benefit the patient by giving pneumothorax on what appears the more likely side. I think I have not known a case in which successful pneumothorax has failed to stop pulmonary hemorrhage; and even if given in the side on which the hemorrhage did not occur, benefit usually results.

Selective collapse, as has been mentioned, does frequently occur and is a very fortunate occurrence. If the lung is free from adhesions collapse is best over the diseased area, but if the lung is not free, the adhesions are usually over the more diseased area.

It is well to keep in mind that with the appearance of fluid, there is apt to be an uncomfortable, febrile period, with nausea and perhaps even vomiting, and the patient will need all the encouragement one can give during that period. It is well to realize that pretty much the same symptoms as those of acute pleurisy will occur in these cases. One can assure the patients that they won't last very long, and that no definite harm is likely to result.

The work of Sangeman is well known, his review of a great many cases with and without fluid, and his estimate that the difference between cases in which fluid forms and in which fluid does not form amounts to a little less than 2 per cent. So the patients can be assured that likely no great damage will result from the appearance of fluid; and that is fortunate, for fluid appears, I think, in about 50 per cent. of cases.

One very important question for discussion in connection with pneumothorax is as to when and how to stop it. We all know more about getting into it than we do about getting out of it. We all know the indications for beginning far better than the indications for stopping. I am coming around to believe that, in many cases, indeed perhaps in most, it should not be stopped. If it is doing the business, helping the patient very greatly, why stop it, unless to make collapse more permanent by thoracoplasty? But when the lesion has been favorable or slight or of rapid type collapse can, I think, very well be discontinued after two or three years, or even earlier.

When extensive advanced disease, however, has been the reason for collapse, it seems wiser to keep going or to substitute thoracoplasty unless, as frequently happens, the pneumothorax brings itself to an end by changes in lung and pleura. In case of bilateral disease, it is of benefit to collapse one side, if the other side is not too badly affected; and if, during the collapse of one side disease spreads in the other lung, it is the part of wisdom to keep on with the collapse rather than to stop it, because by pneumothorax one is cutting off a certain amount of toxemia and so benefiting the patient.

The question of pleurisy came up. In a tuberculous case, one sometimes draws off fluid, substitutes air, and continues the pneumothorax. That is wise, only if the lung needs that amount of collapse. If it is a tuberculous lung and needs to be collapsed, that is a good way to introduce pneumothorax and one should keep it up on account of the condition of the tuberculous lung. I doubt the wisdom, however, of using pneumothorax and the putting in of air in ordinary cases of pleurisy. I may or may not be right, but my observation has been that a pleurisy which clears up with fluid remaining pretty much in the chest gives a diaphragm in much better position than a pleurisy cleared up by the putting in of air. That is, when it is left pretty much to Nature's method, the diaphragm is smoother and has less adhesions to the wall than

otherwise. The putting air into an ordinary pleuritic space, on account of ordinary pleurisy, is undue intervention, and is apt to lead to abnormal adhesions of the diaphragm.

Pneumothorax for diagnosis is particularly useful, I think, in septic cases where the foci are small. One knows one has a septic condition of the lung, perhaps, following aspiration after operation, and yet one cannot detect any very distinct focus. One will be able to detect that focus very much better if one does a pneumothorax for diagnostic purposes.

Pneumothorax is distinctly of use as treatment in these septic cases in which the foci are small. Sometimes even when symptoms have extended over a great number of years, much can be accomplished by a pneumothorax. I have two cases at the present time in which cough and expectoration on account of small basal bronchiectasis has lasted for many years and in which a small pneumothorax has improved all symptoms and the general condition.

There is one problem or phenomenon in connection with pneumothorax that I have not heard mentioned. It occurred to me that the interesting work reported by Dr. Bettman might perhaps lead to some solution of it. In a number of cases in which pneumothorax has been kept up for some considerable time, in which there has been fluid, and in which, undoubtedly, there have been adhesions, the occasional filling with air is followed by a reaction in which the patient has considerable temperature for several days, and sometimes nausea follows. These fills do not need to be large; it doesn't matter whether they are large or small. They occur in patients who have had pneumothorax for a long time.

One great interest in pneumothorax, it seems to me, to a physician in an association of thoracic surgeons is that artificial pneumothorax is the training ground for thoracoplasty. At least, what we have learned in connection with artificial pneumothorax might very well be applied, in part at least, to indications to thoracoplasty, especially in discussing indications and contraindications. It seems to me that one could come best to the selection of cases for thoracoplasty very well indeed through a training in pneumothorax.

Dr. Franz Torek, New York: The clinical classification of cases, as presented here, has been correctly made as incipient, moderately advanced and advanced cases. Now, while this is good for clinical purposes, a much better classification for practical purposes is the pathologic one into the productive and the exudative variety. We know that the productive variety, which means the cases in which granulations are produced, are the ones that are favorable for surgical intervention, whereas the exudative variety, in which there is an intercanalicular process with a free exudate, for instance, the lobular, cheesy and the lobar cheesy variety, are not favorable for surgical intervention, as they represent a condition in which the body shows little reaction in the way of defense.

Now, how can we tell the one variety from the other? In the first place, by the history; the exudative variety has a short history; the productive variety often has a history extending over a year or several years.

The productive variety usually is accompanied by little or no fever, whereas the exudative variety is accompanied by fever, sometimes very high fever.

The roentgenogram gives valuable information. The productive variety shows itself in the form of comparatively small nodes or clusters of nodes, say, for instance, like the shape of a clover leaf, each single node showing a definite infiltrated margin; whereas the exudative variety shows shadows of the thinner variety without a thick margin, often appearing as if they

had been washed out, one flowing into the other, the characteristic appearance. It is perhaps best shown in the roentgenograms of patients with cavities. The cavities in the productive variety have a well marked outline or frame around the cavity, whereas a cavity in the exudative variety is like a punch hole without any frame. Therefore, we often can properly distinguish the anatomic-pathologic form from the roentgenogram.

We have heard here that a number of cases of pneumothorax have failed because of the presence of numerous adhesions. These kinds of cases are the most favorable for surgical intervention because it is the productive variety that form fibrous tissue, which forms the greatest number of adhesions; consequently, these cases which the pneumothorax cannot influence are benefited by surgical intervention.

On the other hand, it is wrong to try to do a surgical operation in a case of the exudative variety because the lung in that class of cases will not collapse to the degree expected from the operation. In that particular kind of case, the pneumothorax is the method of treatment, because at the time one introduces perhaps only 100 or 200, or at the utmost, 300 c.c. of air, which is about all that that lung is capable of collapsing. One who has ever opened the chest and seen the exudative variety, as I did in one case, can see that such a lung does not collapse properly; it stays almost the original size, like a pneumonic lung.

So much, then, for the differentiation and classification which I much prefer to the clinical classification of incipient, moderately advanced and far advanced.

The matter of pleuritic exudate following pneumothorax has been touched, and the question raised as to what accounts for it. My own belief, in this matter, is that we have to deal not with pleurisy but with a transudate. In these cases of productive tuberculosis with formations of fibrous tissue, there is a pull exerted by these fibrous formations in the direction toward the hilum and a pull in the other direction by pleuritic adhesions, while reabsorption of part of the injected gas results in a strong negative pressure. In that way, there is a certain amount of suction on the surface of the lung. The fluid, therefore, is being sucked out of the lung; it is a transudate, to my idea, and not an exudate, and that very simply explains the presence of the serum in the pleural cavity in the majority of these cases.

Dr. Lemon made a remark about toxic material being squeezed out through the lymphatics at the time of the collapse. In my opinion, that is not quite correct. If the lung is collapsed, the lymphatics are tightened up and partly blocked, and there will be less absorption through the lymphatics after the collapse than there was before. In fact, a great many of the most remarkable improvements in the first days after pneumothorax are due to the fact that the absorption from the lymphatics has been stopped by their compression. But there is another thing that occurs in cases in which pneumothorax has been applied. Wherever there is a disease, there are formed antibodies and antitoxins; so these also will be found in the blood of the tuberculous lung. These toxins act in a similar manner to tuberculin, which, if given in excess, causes severe exacerbation in the patient's illness, and when the lung collapses, they are driven into the general circulation.

In producing a collapse we do two things: we block the lymphatics, but we also compress the vessels. Now, in the blood vessels there is present this toxin. When the lung is compressed, this toxin is thrown into the circulation, and, for a time, will make the patient sicker. On the other hand, the compression of the lymphatics will prevent absorption and make him feel better.

The question whether he is going to feel sicker or better after the pneumothorax depends on whether he has more of the toxins pressed into the system through the vascular system or whether he has less toxin absorbed, owing to the compression of the lymphatics.

DR. CARL A. HEDBLOM, Rochester, Minn.: The question has been raised as to the treatment of the purulent effusions. We see a comparatively large number of cases of tuberculous empyema here; about 20 per cent. of the cases that come to the clinic are tuberculous. Clinically, they might be classified as those that give a history of having had a so-called idiopathic purulent effusion without any evidence of tuberculosis. In the second group are cases that show either an old lesion apparently healed on one or other apex of the lung, or a tuberculous kidney, or tuberculosis somewhere else in the body; and in the third group, the patients are frankly tuberculous, with an active lesion, usually with bacilli in the sputum.

We make the positive diagnoses of these cases as being tuberculous on the microscopic findings, the finding of a characteristic histologic picture in the excised pleura, or the finding of the bacilli in the exudate, or by guinea-pig inoculations, so there is no question as to these cases being tuberculosis.

There also is another classification, that is, those who have undrained cavities and those who come with cavities that have been drained. Most of the patients we see have been drained. Their cases have been, as a rule, mistaken for ordinary empyema, and they have been drained; at least, we judge that from the history; and then they come to us with a double tuberculous and pyogenic infection.

The treatment depends a little, of course, on the clinical aspects of the case; one wouldn't treat a patient with a small cavity with the pyogenic infection as the primary condition in quite the same way as one would treat the massive collapse of the whole lung.

I take it that the type that we are particularly interested in here is that in which the lung is about completely collapsed and there is a mixed infection. That type, of course, occurs in the undrained case in which there is a perforation of a bronchus.

We feel that it is a great mistake to drain by the open method a sterile tuberculous effusion, even though it is purulent, because this leads to secondary infection. In cases that are secondarily infected, as proved by culture of the aspirated effusion or by the presence of a fistula, one has to deal with the pyogenic infection, more or less as though the tuberculous condition were not present, and one has to follow the same principle of treatment in those cases as in a case of the ordinary nontuberculous type. We try to use the irrigation treatment in order to clean up the pyogenic infection, to improve the condition of the patient and to see what we can do in the way of expansion of the lung, in cases in which there is no evidence of active tuberculosis.

We like to use surgical solution of chlorinated soda (Dakin's solution) in the case in which there is evidence of thickened pleura protecting the lung. In the cases in which the roentgenogram shows a translucent chest, if one can use that term, it has been our experience that it is not well to use surgical solution of chlorinated soda because of the probability of foci being present near the surface of the lung and the solution corroding the surface and making an opening into the lung and producing hemorrhage. We have had patients harmed by using surgical solution of chlorinated soda under those conditions.

After one has done all that is possible in the way of preparation for operation, the treatment is a collapse of the cavity by an extrapleural thoracoplasty;

that is the operation of choice. We have had cases here that have run the clinical course typical of an active tuberculosis process, in which, after the cavity has been cleaned up, we have carried out practically the same operative procedure that we use for collapsing the bronchiectatic lung. We have succeeded in obliterating cavities which involved the whole pleural cavity, including the dome.

It requires, according to my experience, a good deal of time, and if one is willing to go slowly and do only as much as the patient will stand, or a little bit less, at each sitting, a great deal can be accomplished.

One patient of that type, one of the last we had, was a man who had a completely collapsed lung and an active tuberculosis. In the course of a few months, he left the hospital with the cavity entirely collapsed; within two months after he left, he was married, and I have just recently gotten a card from him announcing the arrival of his first born. He is a young doctor, and, of course, that kind of result encourages one to think that even a long period of time and infinite pains is worth while.

I think that, as a general rule, that type of treatment promises the most. I don't believe that one can cure all these cases, but that general method of handling the cases certainly has produced gratifying results, and, during the last years, we have not had any operative mortality in these cases of tuberculous empyema.

Dr. Sidney F. Blanchet, Saranac Lake, N. Y.: I have been rather conservative in using artificial pneumothorax for the control of pulmonary hemorrhage. If the bleeding is small and responds to ordinary measures, I do not attempt to induce artificial pneumothorax because of the possible presence of adhesions which may increase instead of control the hemoptysis. When I do employ this measure, I am content to introduce a moderate amount of gas; being prepared to run back to the patient to introduce more or to withdraw some according to the results secured. I believe that it is risky to attempt to use artificial pneumothorax if one isn't reasonably sure from which lung the blood is coming.

In regard to the stage of the disease to treat by this method, in my opinion, it is very unwise to induce artificial pneumothorax in early stages of pulmonary tuberculosis in which good results can be expected from the usual sanatorium treatment. If one does use it early and this patient should relapse later on, the lung usually will be found so firmly adherent to the chest wall that this treatment cannot be employed at this time when the indications for its use are comparatively urgent.

Of course in those cases which come to us when the disease is advanced, there is little choice; but many of these cases would have been ideal for artificial pneumothorax treatment if they had come for consideration earlier. In moderately advanced cases that do not respond reasonably soon to ordinary treatment (in which the indications for the use of artificial pneumothorax are ideal), I believe experience has shown that the best results are secured.

In starting my compression, I always have gone slowly, using small doses, according to manometer indications, and also depending on the amount of pain produced. This secures a gradual, careful compression of the diseased lung and, also, as the adhesions are forced apart, a minimum amount of pain. Occasionally, either with or without a cough, a rupture of the lung occurs, possibly as the adherent part of the lung is being separated from the chest wall.

In regard to effusions, I have come to be content in most cases to leave them alone. As a rule, the period of reaction is short and, in most cases, especially

in the moderately advanced group, these effusions are more or less temporary, becoming absorbed sooner or later.

I agree with Dr. Torek that when the compression is slowly induced there is not much unfavorable reaction, that is, there is less absorption through the lymphatics. The response of the patient usually is fairly prompt, especially in the group in which the indications for its use are favorable. I was interested in Dr. Torek's urging a pathologic classification.

As he said, those cases in which adhesions prevent the use of this method probably belong mostly to the productive type; while the others, in which a good compression is secured, fall into the exudative group.

Dr. J. J. Singer, St. Louis: I would like to discuss the word "percentage." Dr. Lemon stated that about 1 per cent. of the cases might be benefited. One per cent. is a small number, but when you stop to think of the millions of people with tuberculosis, 1 per cent. is a great deal in numbers. If we have benefited 1 per cent., we have done a great deal, and it is worth the effort.

The important message that pneumothorax has for us here, and the world over, is, I believe, that a patient with tuberculosis who isn't doing well should be put to bed and at rest; if that doesn't help, pneumothorax should be attempted. If that doesn't help, thoracoplasty; if that doesn't help, I don't know what to suggest.

Pneumothorax is a diagnostic device, you might say, for selecting the kind of case suitable for thoracoplasty; it is the purpose of this association to have men who will do pneumothorax work, to prepare our patients for the so-called last resort.

I have hopes that thoracoplasty will not always be the last resort. If one has only that kind of patient, one will probably get bad results, as one does with so many other patients who come only when perfectly hopeless.

A CONTRIBUTION TO THE SUBJECT OF EXTRA-PLEURAL THORACOPLASTY IN THE TREATMENT OF PULMONARY TUBERCULOSIS

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In speaking before a society of this sort, I feel that it is unnecessary to go into any detail on those points in the operative treatment of pulmonary tuberculosis which are well established, and must be familiar to all. The standard posterior thoracoplasty, as adopted by Sauerbruch, Bull and others, is recognized nowadays as being a classical operation, applicable to the majority of cases. I shall, therefore, with regard to such general considerations as the selection of cases, the operative technic, anesthesia and after-care, confine my remarks to a few paragraphs embodying my own views, and shall then proceed to discuss certain particular types of operation, and conclude with a survey of my own results.

Beginning, then, with that matter of prime importance, the selection of cases, it is perhaps hardly necessary to say that close collaboration with the expert in tuberculosis is essential. This point need not be labored. The general contention that the opposite lung must be practically sound, a contention so much insisted on by Sauerbruch, seems to me to be somewhat rigid. In none of my cases has this ideal condition of affairs been present. In all, there has been a certain amount of disease in the other lung, and, in a few instances, this has been active. I feel that, if we narrow too greatly the indications in this respect, we shall have to refuse operation to many who would be benefited by it. Still, one has to be cautious; and one is cautious in the same way as when deciding to institute a pneumothorax under like conditions. Thus, one should not do too much at once. The rule that I have followed, with but one exception, has been to operate in not less than two stages. In two instances, indeed, the operation was divided into three stages, both patients coming through perfectly well. One can also reduce the danger by taking care not to remove an undue length of rib, a point to which further reference will be made.

I have not yet felt the necessity of adopting Sauerbruch's preliminary phrenicotomy as a test of the soundness of the other lung, although I anticipate taking advantage of it on occasion. As an independent measure, it appears to me to be indicated rarely. For one thing, the pathologic condition of a predominating lower lobe lesion, for which phrenicotomy is particularly advised, is rarely present;

furthermore, a careful thoracoplasty carries with it a very slight risk of aggravating a lesion in the other lung; and, finally, due consideration of the course of the disease, with particular regard to making an accurate estimate of the patient's general resistance, will nearly always suffice to decide the question.

Concerning the value of phrenicotomy as a mode of treatment, whether as an independent procedure or as an aid to thoracoplasty, I can offer no opinion of value, as I have only once performed it, and in this case the result was quite indecisive.

The key note in the selection of cases lies in a correct estimate of the patient's resistance. In that sense, the type of case suitable for the operation remains, and must remain, the chronic fibrotic, with cavitation and a good deal of shrinkage. Fibrosis and scar contraction are the outward and visible signs of resistance. Yet I have come to think that two other types may reasonably be included. Of these, one is characterized by very extensive fibrosis with consolidation, and with very little tendency to destruction. The pleura is thick; sputum is small in amount, and may contain very few bacilli; there has been widespread and probably acute exudation throughout the parenchyma at some previous time, but the process has been halted, much exudate has been reabsorbed, and much fibrosis has occurred. The roentgen-ray examination shows a uniformly dense shadow over the whole or greater part of the lung, and there is no detail to be seen. Such a patient does not stand exercise; he is still on the edge; and one gets the impression that any carelessness in treatment would result in rapid and perhaps extensive destruction. Pneumothorax is impossible, but a thoracoplasty, putting the lung definitely at rest, should safeguard him, and allow an earlier and safer resumption of exercise, and a much greater prospect of definite arrest. One patient of this type was referred to me by Dr. Parfitt of Gravenhurst (Ontario), to whose refinement of judgment we owe the establishment of this particular set of operative indications. His patient, a young man of about 20, on whom a posterior thoracoplasty was done a year ago, has been very markedly improved.

The second type is that in which one finds a more or less rapid progression of active disease of the exudative form throughout one lung, threatening shortly extensive cavitation. Sauerbruch, in his first article, in 1913, called attention to this class, and reported two cases of very marked improvement following thoracoplasty. This type, I believe, is currently accepted by the experts as being suitable for pneumothorax. I have had only one patient of this sort, and he did well for some eight months, but died at the end of eighteen months from involvement of the other lung and the intestinal tract. I am inclined to think that a preliminary phrenicotomy would be advisable in this class.

TECHNIC

With regard to technic, I shall do no more than express briefly my own views. As already stated, I adhere to the two stage principle. In forty-five cases of the standard posterior rib resection—that is when the operation was confined to a posterior thoracoplasty—there is no death to record before the twelfth day after operation. This patient was in poor condition. He was operated on in a distant city, and was not under my care from the second day on. His physician reported that he died of cardiac failure. I had resected from the tenth to the fifth ribs inclusive, leaving the second stage to be done later by the local surgeon. It was noticed during the progress of the operation that his lower lobe was not well supported by fibrosis, and sank in a good deal during inspiration. It is possible that I took out too great a length of rib, on the average from 5 to 6 inches (12 to 15 cm.). I shall speak later of the results in detail, but I note this death here as being the only one in these forty-five cases immediately attributable to the operation per se, that is from shock, or from respiratory or cardiac failure.

I have only once operated in one stage, and that patient did well. This point is interesting in that the continental operators, as I find from the literature, who at first all favored one stage as a general rule, are veering round very strongly toward the two stage procedure. Their operative mortality, with the exception of that of Sauerbruch in his later reports, is much higher than the figures that I am able to report; and I believe that their turn toward the two stage operation is the result of their finding that the one stage gives the higher mortality. I have, indeed, come to believe that the standard posterior thoracoplasty, in two stages, involves very little more risk to the patient than an artificial pneumothorax, and I approach it with confidence.

There is another factor of safety which demands an emphatic word. The length of rib to be resected should not be judged by rule of thumb or by unvarying rule. That one authority advises the removal of from 4 to 8 cm., while another emphasizes the importance of resecting from 6 to 18 cm., signifies nothing. To my mind, both are right and both are wrong, according to circumstances. In general, I believe that Sauerbruch goes too far when he restricts his resection to from 4 to 8 cm. Bull and others remove a much greater length of rib. But I note that Sauerbruch's immediate mortality is the lowest of all. In fact, the operation in his hands is very safe, whereas Bull and others have to report a much higher operative mortality. Accurate judgment in this matter should, I take it, be based on an estimate of the patient's ability to withstand the operation. In that sense, the length of ribs to be resected should depend on the amount of disease, the degree of insinking of the lung on inspiration during the course of rib removal

and also on the general resistance of the patient. If the lung is fairly stiff, one can resect safely 16 or even 18 cm. of rib; and on the other hand, if the lung sinks in considerably during inspiration, one should remove a much shorter length. A lung which is diffusely diseased and fibrotic shows usually very little insinking. The lung is held out by its own mass, and consequently the danger of mediastinal flutter is to a large extent reduced. But if it does sink in seriously during inspiration, there immediately appears paradoxic respiration, and, in such cases, the resection of too great a length of rib exposes the patient to the danger of mediastinal flapping and gradual failure of the heart. I feel that in one case I did resect too great a length of rib, with consequent death of the patient. As an average, I resect from 5 or 6 up to 14 or 15 cm. of rib, and, on the whole, I have found that that length represents a good middle ground. As a matter of fact, Sauerbruch's "practical cures," as he terms them, are somewhat lower than those of Bull, and I think that the difference lies in this: that the greater length of rib one resects, the greater the chance of a permanent and satisfactory arrest of the disease, but also the greater the immediate risk of death from operation. In one German report from Davos (Stöcklin) embracing 100 cases, I found the quite prohibitive operative mortality of 20 per cent.

Now, as to the anesthetic, that, of course, is a permanent bone of contention. I have never been willing to use local anesthesia alone; nor to use ether, and much less chloroform. The objections are fairly well known. For my own part, gas-oxygen with a local anesthetic, in addition, seems to me to be the ideal anesthetic, because one needs not merely anesthesia as regards pain, but also anesthesia as regards the mental distress which operation usually causes in these patients. I confess, however, that when the amount of sputum is very large I am inclined occasionally to use a local anesthetic alone. Nevertheless, by dint of bringing on the operation after the patient has emptied the lung by proper coughing, I have got through quite well with gas-oxygen, without any particular ill results. The objection, of course, to gas-oxygen is that, during anesthesia, one may get an aspiration infection in the good lung or in other parts of the diseased lung. In my experience, that has occurred only once, and I feel that the danger is so slight that I prefer to go on with gas-oxygen anesthesia for the sake of the greater comfort of the patient. Indeed, local anesthesia itself is not without its danger. One may have to use so much of the procain as to render the patient very miserable with vomiting for some time after the operation; and Willy Meyer, I believe, considers that one of his patients in whom he carried out paravertebral anesthesia died from the effect of the anesthetic itself. I note also, in the continental literature, a very great tendency to give up local anesthesia alone in

favor of a general anesthetic. Sauerbruch is using ether again; and Bull is using a mixture of ether and chloroform, three parts to one. They have found, obviously, that a local anesthetic alone is not the ideal, and they are now saying that the danger of a well given general anesthetic is not so great as they thought, and apparently is counterbalanced by the greater speed of operation and the comfort of the patient. Ethylene may prove to be still better than gas-oxygen, but I have not yet used it. In a few cases, indeed, I have omitted the local anesthetic, and found that the patient got along very well, and, as a matter of fact, I am omitting procain more and more whenever the gas seems sufficient. The patients do not seem to suffer appreciably from shock; and I have got the impression that post-operative vomiting is much less when procain is omitted. I have given up alcohol injections in the intercostal nerves. It seemed to me to cause neuritis, and it delays the operation.

OTHER PROBLEMS

I proceed now to discuss more or less briefly certain other problems of interest.

First, the problem of the large apical cavity, the rest of the lung being fairly safe, either from there being not much disease in it, or from its being compressed by a previous thoracoplasty. When a patient has a large, old cavity and very little sputum, containing comparatively few bacilli or even none—in other words, when the patient has an excellent chronic case, with good prospects, and he is able to do some work, I feel that the outlook for obliterating such a large cavity by operation is so poor that it is hardly worth while undertaking a big operation for that purpose. Of course, some cavities, even fairly large, are obliterated by the ordinary posterior thoracoplasty, and I recollect several such examples. But quite often the cavity remains only partly compressed; and, if there is any danger from it, that danger persists as much after as before the thoracoplasty. The question in such cases is, Should one do an apicolysis, or should one abstain from any operation? On the whole, in such good cases I am in favor of abstaining. On the other hand, when the cavity is discharging a considerable amount of purulent sputum, with large numbers of bacilli, it becomes more worth while to attempt its obliteration. The first thing to do, of course, is a proper posterior thoracoplasty. That may occasionally succeed. If that fails, the question arises whether one should proceed to do an apicolysis, and fill the space created with fat, paraffin or muscle. In my hands, a thorough apicolysis, if the rest of the lung is in an advanced stage of disease, has proved to be a dangerous operation. Otherwise, that is, the rest of the lung being in pretty fair condition, it has not been dangerous. I think the distinction lies in the general condition

of the patient. In old, advanced, toxic cases with great destruction, the operation is dangerous. In good chronic cases, it is not. Of course, one may ask, Why not operate from the first to compress such an apical cavity, if the rest of the lung is in good condition, and omit the posterior thoracoplasty altogether, doing merely an apicolysis? That, as a matter of fact, was the operation of apicolysis as first advised by Tuffier, and its purpose was to avoid doing any extensive operation on the rest of the chest wall in incipient cases. This also was Baer's idea with his paraffin filling. But nobody nowadays advises the operation for incipient cases. For advanced disease, localized to the apex, it may be suitable. My experience has not yet embraced a case of this kind. The lower lobe has always shown a considerable amount of disease, and so much so that a thoracoplasty was indicated from bottom to top. Yet I can easily see that a few cases might be considered suitable for this localized operation; it is a limited operation for a limited disease; and, for such patients, I am feeling more and more inclined to do an apicolysis only. But when at all in doubt, I should prefer to err on the side of safety, and do, first, at least, the complete posterior thoracoplasty, for disease in the lower lobe may be much more extensive than is discoverable by physical signs, or even by roentgen-ray examination, and there is always the danger of aspiration into the lower lobe if one compresses an apical cavity without previously having compressed the lower lobe. In any such case, however, that is, omitting the posterior thoracoplasty, I should be inclined to do a phrenicotomy first, in order to guard against the danger of aspiration.

There is, secondly, the question of doing a partial thoracoplasty to supplement a partial pneumothorax. I have done this in four cases, and am no great friend of the operation. I find certain disadvantages in leaving a partial pneumothorax. There is, to begin with, the tendency to adhesion and to gradual loss of the pneumothorax, in which case that portion of the lung expands and is exposed to fresh danger of infection. In the second place there is some danger of a pneumothorax of this sort becoming infected by a perforation from a lung cavity, and the development, consequently, of an acute pyopneumothorax, which will carry the patient off, as happened in one of my cases. This patient had an upper pneumothorax, and the lower lobe was compressed by a thoracoplasty from the tenth to the fifth ribs. Within a month, a peripheral cavity had broken into his pneumothorax, and he died of the acute infection. I thought that the increased pressure induced by the thoracoplasty, acting on the pneumothorax, might possibly have favored the perforation of a mural abscess into that pneumothorax; and probably under such conditions, one ought to remove part of the air in the pneumothorax in order to avoid any possible increase of tension.

There is, further, the disadvantage arising from the need of constant refills, which have sometimes to be carried out by the local physician at home, who has no conception of the method, and frequently refuses to undertake it. Under these circumstances the pneumothorax lapses, the lung expands, and then there is apt to be trouble. Consequently, I am in favor of giving up a partial pneumothorax and doing the complete thoracoplasty. This argument applies also to subtotal pneumothorax, though to a less extent.

There is one set of circumstances in the presence of which I feel that we should substitute thoracoplasty for pneumothorax; and these are, briefly, the occurrence, and recurrence after repeated aspiration, of fluid in the pneumothorax cavity, particularly if the fluid shows tubercle bacilli, or if it is frankly purulent, even if organisms cannot be demonstrated. I believe that such cases are constantly exposed to the danger of suppuration, whether by increase of virulence of organisms embedded in the pleura, or by frank perforation. The consequent pyothorax is apt to carry the patient off. The only safeguard against such an accident is to get pleura to pleura, and the only way to do that is to do a thoracoplasty from the eleventh up to the first rib. In two cases in which frankly purulent fluid had reaccumulated, and had persisted from one to two years, although the fluid in both was sterile, I performed a complete thoracoplasty, and both patients have done very well, the pleural suppuration being entirely abolished.

Pneumothorax, anyhow, according to recent statistics, does not show very brilliant permanent results, and there is always the difficulty of maintaining it. Some believe that, when once a pneumothorax is started, it pretty well has to be permanent. That means a long business. On the whole, I feel that, although pneumothorax is ordinarily to be tried first, a thoracoplasty should be adopted whenever difficulties arise.

A third problem, not at all an infrequent one, is that of the coincidence of chronic intestinal tuberculosis. I recall two patients in whom, at the time of the operation of thoracoplasty, chronic intestinal tuberculosis, demonstrated by roentgen-ray examination as well as by clinical symptoms, had been present for months, in one, for over a year. Both these patients seem to be doing well without any aggravation of their intestinal condition. One of them, indeed, was operated on some seven years ago, and represents now one of the "practical cures." In short, when resistance is good, it is obvious that the intestinal disease may be cured by the processes of nature equally with the pulmonary disease, and I regard this condition as not offering a serious contraindication. The same is true of laryngeal tuberculosis. My patients, in several instances, have had this as a complication, and it was cleared up.

Phrenicotomy, preliminary or adjuvant, has caused much discussion of late. Concerning this, I have nothing of value to say. I have done

this operation on one patient, but the result was quite indecisive. I noticed in that patient that the paralysis of the diaphragm was as complete after three months as it was at first. I know that the German literature, in particular, is strong in its favor; but it seems to me that if a patient is reasonably fit for the posterior thoracoplasty, there is no necessity of doing a preliminary phrenicotomy, because the resection of the eleventh rib paralyzes the diaphragm quite sufficiently without that addition. It is true that, when one hesitates in the presence of disease in the good side, and wants, following Sauerbruch, to test out the extra strain put on that good side, one may do a phrenicotomy as a sort of trial. Hitherto, I have not seen that necessity, and with the comparative innocuousness of the posterior thoracoplasty I fancy the necessity will not often arise.

As to the cauterizing of adhesions, according to the method of Jacobaeus, for the purpose of accomplishing a total pneumothorax, I feel impelled to record a strong objection to the method. The danger of hemorrhage is not inconsiderable, and the danger of pyothorax is very definite. In about 10 per cent. of Jacobaeus' cases, empyema resulted; that is, in seven cases out of seventy, and three of these patents died. I submit that an open incision, which allows one to hook up the band of adhesion and ligate and divide it in the wound, is a much safer procedure. This I have done in two cases with excellent results.

There are a few other points, suggested by a reading of my case reports, which I may touch on briefly. Age, for instance, would seem to be no barrier. My oldest patient was 55, and had extensive and severe lesions. He stood the operation quite well, and is now practically cured.

It is remarkable how very favorably some patients who have been suffering for many months from severe toxemia are influenced. Two patients, in particular, stand out in my memory. In one of these, there had been present for at least six months, an elevation of temperature of from 101 to 103 F., together with a pulse constantly over 100, and often 120. Night sweats had been frequent. The amount of sputum measured between 12 and 16 ounces (355 to 473 c.c.) a day. The sputum, after operation, was diminished by 75 per cent., and the cough almost disappeared. Her cyanosis disappeared, giving place to a good color. In short, the change was very remarkable, and I would never hesitate, other things being equal, to operate on patients who have been much run down by prolonged fever and rapid pulse. On the other hand, if the whole lung is very extensively diseased, if cavities are numerous and large, and involve both lobes, whether fibrosis is very marked or not, I consider that, in such cases, the ordinary posterior thoracoplasty may be attempted as a last resort, and with very little fear of an operative mortality; but it may very easily fail of accomplishing sufficient good; and if it does fail, I am against further operation (e. g.,

apicolysis) in an attempt to improve matters. I still think that the operation is worth while, even in these bad cases, as a palliative, because the cough is frequently so much lessened that the patients declare that, for this alone, they would be willing to undergo the operation a second time. Still, one must be cautious with it.

One might ask, what is the danger of infection, either pyogenic or tuberculous, of the very large wound that is made. In reply, I would say that I have had one streptococcus infection, with a fatal result on the twenty-first day. This patient had been twenty years sick, was very septic and was altogether an unsuitable case. It was in 1914, and I believe that at present I would refuse to undertake it. The patient had had fifteen or sixteen punctures in attempts to induce pneumothorax; and one may surmise, not unreasonably, that infection had been drawn out by the needle from the septic lung into the back muscles, and had later been stirred up by the operation. On the other hand, I have had three tuberculous infections of wounds, but they all healed within a few days or a few weeks. On two occasions, I have found a tuberculous gland embedded in the muscles of the posterior wall. I have removed these, and demonstrated their nature by microscopic sections. These patients did not do worse than others in whom they were not found. It is obvious that there is a communication between the pleural lymphatics (the pleura being adherent) and unsuspected lymph glands lying in the back muscles, and that is a point which one must remember.

In one case, I have been obliged to do a second thoracoplasty, the first having accomplished insufficient results. In this patient, I had to remove reformed bone, and also lengths of the ribs running forward as far as the midaxilla. This patient was operated on only some five months ago, but the conditon is improved.

Repeated hemorrhages form a definite indication for operation, failing pneumothorax, and in a number of cases the hemorrhages have been permanently stopped. Nevertheless, I have to report two cases, in one of whom hemorrhages recurred after some six months, and ultimately contributed largely to the patient's death, while in the other the patient's vitality seemed to have been so much lowered by repeated hemorrhages, that he did not stand the operation, though it was only a partial one, and died on the twelfth day.

I come now to a consideration of results, and these are perhaps most usefully presented in tabular form (Tables 1-5). One should premise that statistics of this sort are not to be taken as absolute. The figures must change from year to year. The patient set down today as "much improved" may be dead in six months, and the one who is counted only moderately improved may in the course of a year or two become a "practical cure."

TABLE 1.—*Estimate of Operative Risk Within Three Weeks*

		Deaths from Operation	
Posterior Thoracoplasty, 45 Cases:*			
1. Total posterior thoracoplasty.....	39	0	
2. Partial thoracoplasty, supplementing a pneumothorax	3	0	
3. Partial thoracoplasty	3	2	Cardiac failure twelfth day; Streptococcus abscess twenty-first day
	45	4.1% 2	

* In six of these forty-five, an apicolysis was done at intervals of six weeks to two and one-half years after the posterior thoracoplasty. Of these, three patients died within two weeks. All three had very advanced cavitation with fibrosis throughout the lung. Operation in such cases should be avoided.

TABLE 2.—*Results in Patients Surviving Operation Over Three Weeks (Operation, Thoracoplasty, Total or Partial, or with Apicolysis Added)*

	"Cure"	Greatly Improved	Moderately Improved	Stationary or Worse	Deaths	Cause
More than 1 year, up to 12 years, 27 cases	6	8	2	1	11	Apicolysis..... 3 Tuberculous pneumonia in seventh week..... 1 Progress of disease 7

	Greatly Improved	Moderately Improved	Stationary or Worse	Too Soon to Say	Deaths	Cause
Under 1 year, 16 cases	6	4	1	4	1	Typhoid in seventh week after operation

TABLE 3.—*Deaths Ascribable to Operation (Within First Two Months)*

Type of Operation	Number of Cases	Number of Operations	Deaths	Case, per Cent.	Operation, per Cent.	Cause of Death
Sauerbruch posterior thoracoplasty, including the six with later apicolysis and six partial thoracoplasties	45	85	3	6.6	3.5	1. Tuberculous pneumonia of good side, seven weeks after operation 2. Sepsis, twenty-first day 3. Cardiac failures, twelfth day
Apicolysis, a third operation following the two-stage Sauerbruch posterior thoracoplasty	6	6	3	50	50	1. Acute suppurative bronchitis 2. Flapping mediastinum 3. Gradual cardiac failure (old desperate cases)

TABLE 4.—*Causes of Death*

I. During Hospital Stay
1. Streptococcus abscess under scapula after first stage; twenty-first day (in 1913)
2. Following apicolysis, having previously stood posterior operation well; old cases; extreme fibrosis and cavitation; sixth, fourteenth and fifteenth days after operation; purulent bronchitis; cardiac failure; toxemia
3.
4.
5. Acute tuberculous pneumonia of good side, seven weeks after operation
6. Typhoid fever, after good recovery from thoracoplasty
7. Cardiac failure on twelfth day after operation; poor condition from severe hemorrhages and prolonged toxemia; tenth to fifth ribs resected

II. Later Deaths (All showed initial improvement)
8. Pyothorax, developing in partial pneumothorax one month after operation
9. 18 months after operation
10. 2 years after operation
11. 3 years after operation
12. 2 years after operation
13. 1 year after operation
14. 3 months after operation

All died from progress of disease, usually in other lung, or miliary; two with hemorrhages. All, except one, showed improvement at first

TABLE 5.—*Summary of Fifty-Three Cases Treated*

	Cases
Total posterior thoracoplasty	33
Total posterior thoracoplasty plus apicolysis	6
Partial thoracoplasty (one patient in hospital awaiting second stage)	3
Partial thoracoplasty supplementing a partial pneumothorax	3
Cutting of adhesion through open incision	2
Phrenicotomy	1
Drainage of pyopneumothorax, with severe mixed infection	5
	53

DISCUSSION

DR. WILLY MEYER, New York: It was most instructive for those who have commenced treating advanced pulmonary tuberculosis by means of surgery to watch the gradual evolution of the operation since the time when the late Professor Friedrich, chief of the surgical clinic at Marburg, Germany, on the advice and the instigation of the internist at the same university, Prof. L. Brauer, performed the first extrapleural thoracoplasty, Dec. 11, 1907.

Let me repeat why this operation was done. One had seen that the Forlanini-Murphy method of producing artificial pneumothorax could assist in getting better slowly tuberculosis patients who did not improve by hygienic measures. It produced collapse of the lung and put it at rest; this is the real effect of the artificial pneumothorax. If it was observed that the pneumothorax could not be produced on account of adhesions that had formed between the costal and visceral pleura, in former years the majority of these patients had to die. On careful analysis, Brauer, the internist, advised his surgical colleague Friedrich, when he himself could not introduce the gas into the pleural cavity any longer, to take away the ribs and, in this way, collapse the lung *with the chest wall.*

Without special experience in these cases, Friedrich went ahead according to the original operation of Schede for chronic empyema raising the arm with the covering muscles of the chest, thus exposing all the ribs (Fig. 1). Then, in a comparatively quick way, he cut out the entire length of the ribs from the tenth up to the second inclusive, together with their periosteum and the intercostal muscles. That in itself is, of course, a big operation for a far advanced tuberculous patient. Yet, the immediate results of Friedrich's method, as he brought them over to us at the time of his visit, in 1909, as the guest of the American Surgical Association and the American Medical Association, were not bad. I remember well when he told us of his first patient, the owner of a factory in Belgium, who had been under the care of Brauer for quite a while. He was very, very ill, and was given up by the latter from a medical point of view. Then Friedrich did the operation proposed by Brauer. The patient stood it nicely, and recovered. In one of the last reports of Brauer, it is stated that that man is absolutely cured today; he attends to his business and walks the stairs, sixteen years after the operation.

It was but natural that this initial type of operation should have become modified in the course of years, as many operations, done in new fields, gradually are modified and improved on.

It is most interesting to see how the three men who had been working together in this field at Marburg at that time have been instrumental in developing the operation, each in his own way. A fourth worker was Wilms of Heidelberg, who advanced his own type of operation.

Sauerbruch, who, in 1909, was the first assistant of Friedrich, had watched the procedure and had particularly watched the subsequent dangers confronting the patient. We all know from our Schede operations for chronic empyema that if we take away the entire chest wall on the affected side, minus the skin, some of these patients develop the same symptoms as if we had produced an acute pneumothorax and did not try to fight its serious consequences by means of differential air pressure. It was this observation in particular which induced Sauerbruch to propose his modification. The second principal cause for him was the not infrequent occurrence of an aspiration pneumonia. To meet this,

Fig. 1.—The Brauer-Friedrich incision. This is the original Schede operation, which enables the operator to raise the arm perpendicularly and expose all the ribs from one end to the other.

he advised doing the operation in two or more stages, resecting the lower ribs, from the tenth to the sixth, at the first stage. In 1910, Sauerbruch was called to the University of Zurich, in Switzerland, as full professor of surgery; there he could now follow his personal inclinations and did not have to submit to the wishes of his chief. He used the posterior half of Schede's operation only; that means that he did not run the incision up in front too. Whoever has done it will remember how easily, after having reached the ribs, one can split the very loose connective tissue beneath the scapula and expose a com-

paratively large portion of the ribs. For this act a good sized incision of the muscles and a trained, cooperating assistant, who lifts the scapula and pulls it well aside, are indispensable. Thus, Sauerbruch resected quickly from this posterior incision a good sized piece of the ribs from the tenth up to the first, in one, or, if necessary, in two stages. He called the incision the "hook incision" and the modification "paravertebral resection of the ribs" (Fig. 2).

At the same time, Wilms of Heidelberg had worked out his so-called "columnar resection.". He said, "Why remove the entire length or a large piece of the ribs?" His advice was to cut out a smaller piece in the rear, and, also, in front of as many ribs as we consider best, of course varying the length

Fig. 2.—Sauerbruch's hook incision, which represents posterior half of Schede's incision and permits of pulling scapula aside sufficiently for resection of posterior two thirds of the ribs from the spine forward.

of this piece, and then leave the central portion in place. After this, he advised to compress the chest wall artificially. A number of surgeons have tried his method, among them some of our English friends (Figs. 3 and 4).

The third member of the learned trio was Ludolph Brauer, who had proposed the exact operation of extrapleural thoracoplasty, the name having originally been coined by Dr. Spengler of Davos. In 1910, he had accepted a call to the University of Hamburg, where he is now the director of the Hamburg-Eppendorf Hospital. He there became the successor of the well known Lenhartz, who, as an internist, had always done his own operations on the lung. Brauer took this up. Of all the medical men in the world in high positions, Brauer, as far as I know, is at present the only professor of medicine who operates for diseases of the lung. He, of course, also had done and is still doing the artificial pneumothorax, the collapse of the lung with the help of the injection of gas.

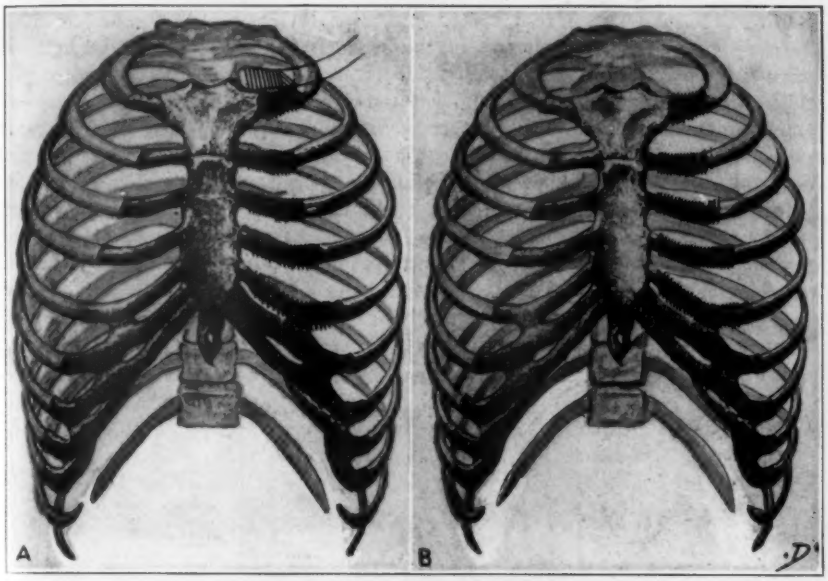

Fig. 3.—Wilms' columnar resection of ribs; *A*, resection of cartilages plus adjacent portions of upper seven ribs, also of sternal extremity of clavicle, in order to increase the contemplated compression of diseased lung; *B*, same operation as in *A*, without the additional operation on the clavicle.

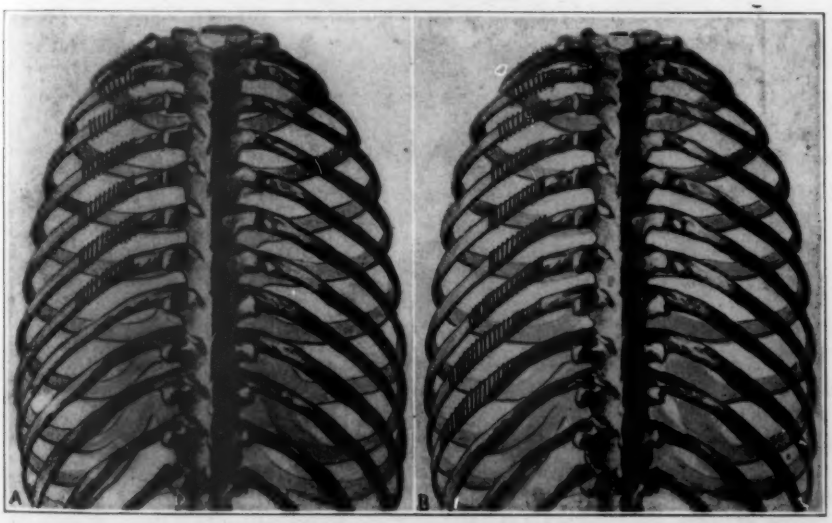

Fig. 4.—*A*, posterior side; several small pieces of upper seven ribs removed in region of their angle; *B*, same operation as in *A*, involving nine upper ribs. Increasing length of resected portion of seventh to ninth ribs should be noted.

On the basis of his personal experience, he calls Sauerbruch's and Wilms' operations insufficient. He terms them "partial thoracoplasty" and considers the excision of the entire length of the ribs, that part which lies beneath the scapula, the operation of choice (Fig. 5). After this is done, he says, the scapula itself can fall inward perfectly and without difficulty. He called his operation, "the subscapular paravertebral resection of the ribs." He favors the one stage operation. These are, then, the three operations in use today for extrapleural thoracoplasty, the original Brauer-Friedrich operation having been given up.

It would, of course, take too much space to go into the details of the three operations. Suffice it to touch a few points. Regarding anesthesia: From what I myself have seen of extrapleural thoracoplasty in cases of advanced bronchiectasis, of which I did seven, and from what I have seen in advanced pulmonary tuberculosis, I am a firm believer in the necessity of not placing the patient under a general anesthetic. We certainly can, nowadays, with the help of procain-epinephrin anesthesia, produce a sufficient local analgesia.

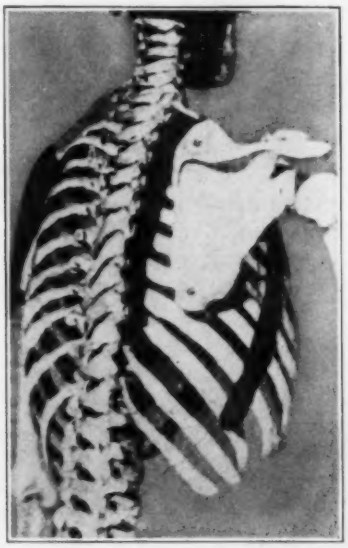

Fig. 5.—Brauer's incisions. He excises the ribs to the very spine; he takes away large pieces on the side of the thorax; in order to do this, he often makes a second incision in front and loosens the periosteum of the ribs in such a way that the rib is pulled out of the periosteal envelop, as we pull the finger out of a kid glove.

Then there is no possibility, at least not during the operation, of an aspiration of mucopus on the part of the patient into hitherto unaffected portions of the lung; and the surgeon is not hurried, a point which Brauer particularly emphasizes. "Aegroti salus prima lex!"

Those American surgeons who visited Sauerbruch's clinic in Zurich just before the World War, in the summer of 1914, were particularly impressed by the excellent team-work at his clinic. Sauerbruch succeeded in reducing the time of the entire operation, from the beginning of the use of the knife until the dressing was applied, to from twenty to twenty-five minutes. Of course, in such rapid operating, it is easily possible that injuries occur to the inter-

costal nerves, a fact which was recently criticized by Brauer. On the basis of clinical experience, Sauerbruch had proposed to resect the intercostal nerves in order to exclude the possible development of neuralgias and traumatic neuritis. Brauer advises not to do that. He says, "I take my time, and I particularly take care that the resection is done very close to the transverse process of the vertebra, and in every instance I preserve not only the intercostal nerves but also the intercostal soft tissues." He also takes care that the attachment of the long muscles in the back to the periosteum of the ribs remains undisturbed, in order to help the quick restoration of the motion of the arm. By doing this, he finds that his patients never complain of subsequent neuralgias.

Of course, it is necessary properly to select the cases, and that is what Dr. Archibald particularly emphasized in his paper. The surgeon must work in close conjunction with the internist. We cannot expect also to be specialists in tuberculosis, but should recognize that the latter understand the segregation of the cases better than we surgeons do, although all surgeons interested in this chapter ought to try to learn it, to be sure.

Friedrich had had the great benefit of having Brauer at his side. Sauerbruch went to Zurich, and got the greater number of his cases from the specialists of Davos, and Brauer, the internist, diagnosed and sifted them for himself and then operated. The specialists at Saranac Lake treat and observe their cases carefully. When they see that medicine and hygiene and the artificial pneumothorax fail to bring further improvement, they segregate and call in the surgeon, frequently Dr. Archibald.

What is segregation? The proper selection of the cases adapted for this operation. It means, for instance, that the diffuse infiltration of the lobe of the lung by tuberculosis, without cavity formation, is not adapted to this type of operation; but, when nature has commenced to form a large amount of connective tissue, when there is cavity formation, usually in the upper lobe, and the lung adheres thoroughly to the chest wall, and when the opposite lung is not affected or only slightly affected, then one has the best case.

It is, of course, necessary properly to prepare the patient for the operation. I will not go into these details because they are a part of the operative work.

To increase the required collapse, we should always include the resection of the first rib. Brauer emphasizes that the hold of the diaphragm is particularly at the eleventh rib. For this reason, he strongly advises against starting resection with that of the eleventh. He says that we should resect from the tenth to the first inclusive, and if, later on, this is found, for some reason or other to be insufficient, we should also remove a portion of the eleventh rib under local anesthesia.

It is of importance to compare the results of the four operators mentioned. The number of Sauerbruch's patients has gone up above 500; Wilms did not live long, he died during the war, and we have not got a large series of cases from him. We also have a comparatively small series from the late Friedrich; a larger one from Brauer. Interesting to state, these four types of operations have had more or less the same final result: between 50 and 60 per cent. of the patients who survived the operation were improved or cured. This means a tremendous progress if we remember that almost every one of these patients, as I said in the beginning of my remarks, had been doomed to die, and that this large percentage was saved by means of the extrapleural thoracoplasty.

Sometimes, the thoracoplasty proves not sufficient. Then phrenicotomy and, eventually, apicolysis are indicated as additional interventions to increase the desired collapse.

The phrenic nerve, as we all know, if resected at the neck, puts at rest the respective half of the diaphragm. Whoever has done it and watched the result of the operation by means of the roentgen-ray examination has seen that the diaphragm really can move up three or four ribs. It, therefore, must be an addition, if indicated later on, in putting the diseased lung at rest.

Sauerbruch has gone so far that he proposes the operation as a prognostic measure; he says that if primarily we resect the phrenic nerve the reaction of the opposite lung will tell us whether the patient can stand extrapleural thoracoplasty or not. Archibald has also emphasized this. From what I have read—not from personal experience—I would say that it seems not advisable to do the resection of the phrenic nerve primarily.

Regarding the operation itself, it has been stated in an excellent paper written by Felix, one of the assistants of Sauerbruch, not long ago, that it is not sufficient "to resect" the phrenic nerve. The phrenic nerve has to be gradually twisted out (exairesis). It seems best, to make the incision posteriorly of the sternocleido muscle, under local anesthesia, go down to its belly, loosen the posterior border, and, with a blunt hook, pull this inward. One then will immediately see the nerve running downward and outward over the scalenus muscles. It is carefully exposed, divided, and the distal end grasped with the forceps. And then, very slowly with one full turn of the forceps in one minute, the nerve is gradually pulled out of the chest, for the reason that, at about the hilum of the lung, additional nerve fibers join the phrenic nerve, which could still innervate the diaphragm. These are usually torn off by this maneuver.

One of the assistants at Schmieden's surgical clinic at Frankfort, Goetze, has written a most interesting article on recurrent hemoptysis of tuberculous patients in the earlier stage of the disease. In a number of instances, he succeeded in stopping it only after this operation. He further considers it a necessity to wind up the period of artificial pneumothorax with this simple operation, which is always done under local anesthesia, in order to avoid a recurrence of the former condition.

It seems worth while to follow up this advice, particularly as we are just in the beginning of this new teaching.

Regarding apicolysis, it was Tuffier of Paris, as Dr. Archibald mentioned, who first proposed it. He used omentum or fat, kept in cold storage, to fill the resulting cavity. Later on, Baer of Davos proposed to use a heteroplastic filling, paraffin of a higher melting point than the temperature of the body. I have done the operation once, and was much impressed by how nicely and easily I could push the apex down. I was not sufficiently cautious, but rather too bold, it being my first operation of this type. When pushing the apex down, I felt a firm adhesion, a band, anteriorly. Instead of being satisfied with what I had accomplished, I wanted to loosen that too. It proved to be the internal mammary plexus. The vein was injured; a furious hemorrhage set in which I could not control otherwise than by putting a gauze tampon into the cavity. After four days, the tampon was removed, and the prepared paraffin plomb, in the shape of small round balls, put in its place; then the cavity was closed completely. Everything went well with the patient for a time. But, after a number of days, fever developed, and I found it necessary to remove the plomb.

Whoever has taken interest in thoracic surgery must have been greatly impressed by its having trespassed on the former absolute domain of internal medicine, the treatment of tuberculosis of the lungs. He must have felt inspired by the fact that, by means of extrapleural thoracoplasty, advanced pulmonary

tuberculosis had become a borderline disease. Among many other colleagues I also was very much inclined to try the effect of the operation in suitable cases. About ten or twelve years ago, I wanted to have such patients admitted to our hospital. But I met absolute opposition on the part of the superintendent as well as of our board of trustees. Of course, I wanted to find out the reason for such peremptory refusal, and learned that we, in the state of New York, have a ruling by the state board of charities that patients with active tubercle bacilli cannot be admitted to our public wards. Whether this ruling is right or wrong, I am not the man to answer. But statistics have shown that in no instance have grown people been infected by being near tuberculous patients with active tuberculosis. This is stated by specialists who have followed up this question with the greatest care and continuous observation. In other words, only the child is infected by tuberculosis, the child's tissues having an inclination to give a hold to the tubercle bacilli.

Now, all these patients who came from sanatoriums, from Saranac Lake or any of those throughout the United States and Canada, have been well trained; they do not expectorate into a piece of gauze or anything they just happen to have at hand; they have a little sputum flask which, when filled, is burned. Into that they deposit their sputum, and so the danger that would occur, even to a younger patient in the adjoining bed, is nil.

It is a fact, generally known and appreciated, that the mortality of tuberculosis has been much reduced by the active campaign led for years by the National Tuberculosis Association. They, too, have stated, I understand, that it is not wise to put a patient with active pulmonary tuberculosis into the general wards of our hospitals. I therefore think that it is not the proper thing on our part to fight this ruling.

A few years ago, I had some interesting correspondence with Dr. Baldwin of Saranac Lake on this very point, because it was impossible for me to overcome the opposition to bringing these patients into the Lenox Hill Hospital. He advised me not to go on with such attempts, but rather to have set aside one room or two in our hospital and to treat the patients there. This certainly seems to be the best way out of the dilemma. But if you set aside a special room in the hospital, you need special nursing, and special nursing is expensive, and there is no question about it that the patients who have undergone extrapleural thoracoplasty need good nursing, certainly in the first days after the operation.

So it came about that not until 1918 did I get my first chance to operate on such a patient in our hospital. It was made possible through the courtesy of our superintendent, who gave me a room for this purpose on one of the upper floors. Fortunately, the patient was able to pay for special nursing. He got well, and I published the case.

The second patient came soon after. He, too, had been sent from Saranac Lake. He was a far advanced patient, and I was not inclined to operate on him; but he begged for an operation, it being his last and only chance. Reluctantly, I did the first stage of the operation under local anesthesia. He stood it well, but soon developed high fever. After some time, he was returned to the mountains under the care of the late Dr. Patterson, who had sent him to me. After a while he died, and the doctor made a postmortem examination; miliary tuberculosis was the immediate cause of death.

I was much interested in hearing and seeing in the charts of Dr. Archibald that he had repeatedly seen death from miliary tuberculosis. It was emphasized

by a former assistant of Sauerbruch that in no instance had they seen exitus from miliary tuberculosis after extrapleural thoracoplasty.

A patient of mine, whose case has interested me intensely, a man, aged 42, had been in bed for over three and a half years. When he came under my care, in October, 1922, he had an almost continuous slight hemoptysis with irregular, high fever; a chronic empyema with fistula; a cold abscess on the back exactly in the line of the required incision; chronic otitis media; also, as Dr. Archibald told us he had found in some of his cases, frequent diarrhea, pointing to a simultaneous affection of the intestinal tract, and both lungs involved in the process, with cavity formation, right and left. An educated man, he saw his only and last help come eventually from surgery. He was ready to take any chance. Soon after admission, the chronic empyema was aspirated; 400 c.c. of thick creamy pus was removed, and then a 5 per cent. emulsion of iodoform glycerin, afterward a solution of 1: 5,000 gentian violet, as advised by Dr. Waters of the Loomis Sanatorium, was injected. The latter fluid proved of the greatest help; it acted in this case almost like magic in reducing the secretion.

For some time after this, I did not know what best to do in this case; in fact, I could not make up my mind to operate at all. Yet I did not want to send the patient home and thus pass final judgment. So, at last, I went ahead. To check the intermittent hemoptysis I started with the resection of the phrenic nerve on the most affected side. Queer to state, the operation arrested the bleeding which had been coming on from every three to four days for the last months.

After a negative aspiration, I split the cold abscess, which contained only tuberculous granulations, packed the cavity, after wetting with iodoform gauze for three days, and then saw it heal, with compression, under one dressing. Then I did the complete resection of the left ribs from the tenth to the first in two stages under local anesthesia.

Soon the patient began to improve. The principal operations were done in January and February, 1923, and the patient stayed at the hospital until March. Then came the question of what was best to do for him. In fact, what shall we do with any of these patients after they have been successfully operated on, if the other side is also affected? In the first patient I operated on, both sides had been affected; he got entirely well. After his two operations, he first went to Arizona, where he stayed for almost a year; from there he went to Texas, taking a prolonged sun bath every day.

Knowing that heliotherapy is of great help in the treatment of tuberculosis, that the sun rays are, in fact, the best means to bring these patients round (real or artificial sun) if anything can be done at all, I also advised this patient to go down South, taking his wife and daughter. We tried to get the required money for him. But he was a child of the North; he had come from Norway, and insisted that he could not stand great heat. So, reluctantly, we could not do otherwise than send him back to the mountains from where he had come. There hemoptysis soon returned, this time, I believe, from the opposite side. He had to be put to bed, in his room, not in the sun. After a number of months he died from increased bleeding from the lungs and persistent dyspnea.

Of course, I cannot prove that this particular patient would have improved had he gone South. However, I believe, it might have been so. Certainly, this point must be thoroughly discussed in the future, whether patients with

bilateral tuberculous affection should be advised, after the extrapleural thoracoplasty, to return to the mountains here in the North and there, if hemorrhage returns, be put to bed in a room, or whether they should go South and be in the open and in the sun as much as possible. I personally think heliotherapy in a warm and dry climate gives these patients the best chance for ultimate recovery.

Dr. Ethan F. Butler, Sayre, Pa.: Resection of the clavicle may aid in obliterating an apical cavity. After removal of the upper ribs, the clavicle alone remains as a bony strut, preventing complete collapse of the upper lobe. I have seen Colonel Keller employ this procedure to advantage in a case of chronic empyema with extensive apical cavity.

The fact that the operation is extrapleural compensates for the relatively large surgical undertaking.

Dr. Leo Eloesser, San Francisco: I think that all of us who have occupied ourselves not only with the surgical but also with the medical side of this affair, and have had the privilege of following tuberculous patients not only surgically but also, more or less, medically, have been struck with the difficulty of prognosis in phthisis. The more one works with it the more hesitant is one in making a prognosis or in asserting that any procedure that one has carried out has really turned the scales in any particular patient's favor.

I am very much afraid to say, after any thoracoplasty, that, had I not operated, such and such a patient would have died; or vice versa, that such a patient will die if not operated on. Too often the patient for whom one prognosticates death, within a few months, will meet one smiling five or six years later, more or less well.

I have been much interested in phrenectomy; one musn't call it phrenicotomy because a simple section is not sufficient; one really has to do as Dr. Meyer says, extract the phrenic nerve and not merely cut it through. The phrenic has several roots, and if one merely severs the main trunk the anastomoses will defeat the effects of section. One has to extract the nerve: the anastomoses are pulled out with it. I have done a phrenectomy on five patients; I did it twice on the indications that Sauerbruch has formulated, as a gauge for the ability of the sounder side to conduct the process of breathing after the bad side is collapsed. Two of these patients went on after phrenectomy as they had before and died in the course of some five months. As they were uninfluenced by phrenectomy, I did not do the Sauerbruch operation.

I have seen hemoptysis occur after a phrenectomy, and I should like to ask Dr. Archibald and others who have had experience with this operation, whether they have seen hemoptysis occur after phrenectomy rather than be stopped by it. On one patient, a dental student who was bleeding profusely, I did a phrenectomy, and that did stop his bleeding for about a week. After a week, he began to bleed again as profusely as before; he bled alarmingly. With my heart in my mouth, I then did a complete Sauerbruch operation; the bleeding stopped; he has gone North, has done well, and is fat and healthy.

As for indications, I think that Dr. Singer formulated the statement that "one puts these patients to bed; if that doesn't help, one does a pneumothorax; and if that doesn't help, then one does a thoracoplasty." I am afraid that if one allows oneself to be persuaded to do operations on the indication of not being able to help the patient by medical means, one will very soon find oneself in hot water. One mustn't operate on that indication. Doctors send patients to surgeons, saying: "I can't help this man; you have to operate on him." But because the medical man can't help him is no reason for one to operate.

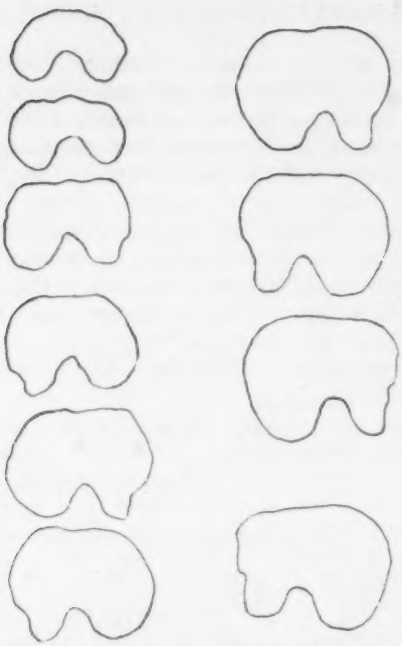

Fig. 1.—Transverse sections of chest after Sauerbruch operation without resection of first rib; collapse is incomplete, the apex not being collapsed at all.

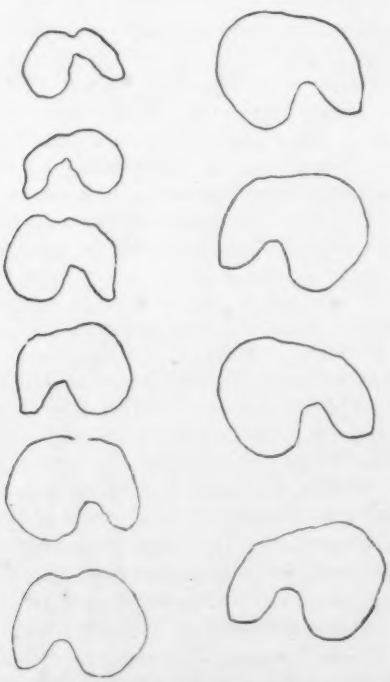

Fig. 2.—Transverse sections of chest after Sauerbruch operation with resection of first rib; collapse much greater than in Figure 1, the apex being especially well collapsed.

One must operate only when one sees a reasonable prospect of doing good.

I think, like Dr. Archibald, that if all of us could see adhesions with the eye instead of by the roentgen-ray examination, we should be afraid of poking an instrument through a small hole and trying to sever adhesions with a cautery. When thick adhesive strands are severed with a cautery, they bleed profusely. These strands are not fibrous tissue composed of adhesions alone; there is lung in them; and, very often, as Dr. Archibald stated, there is in them a small, drawn out cavity. Now, if one burns those things through without ligating them, one is going to get into trouble. The man's lung is going to blow out into his chest. There are not only thin strands of adhesions, but sometimes there are broad sheets; it is manifestly impossible to separate these sheets with anything like a cystoscope or a cystoscopic cautery. You have to sever them with a knife, or pull them off from the chest wall together with the visceral pleura, and that is no job to do through a small keyhole.

My statistics are: Sauerbruch performed twenty operations on sixteen patients; of the sixteen, three are dead. One died three years, one six months,

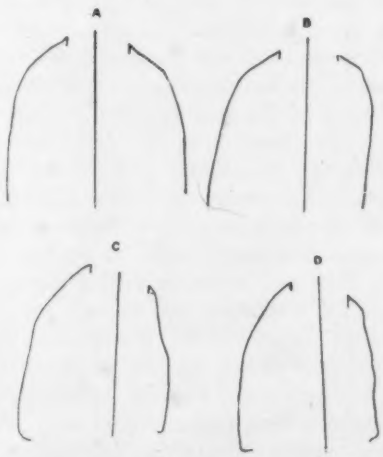

Fig. 3.—Frontal section of chest: *A*, normal; *B*, Sauerbruch operation without resection of first rib; apex remains unchanged; *C*, Sauerbruch operation with resection of first rib. The apex is entirely collapsed: the operated side is not only narrower in the transverse diameter but it is also shorter in the vertical one. The apex has dropped several intercostal spaces; this drop is especially important in effecting the apical collapse; it is wanting if the first rib is not resected. *D*, same as *C*, but with manual compression of resected side simulating the compression produced by recumbency on the affected side and by the normal drag of the viscera and abdominal muscles on the chest wall when the patient is in an upright posture. The vertical drop of the affected side is increased over that seen in *C*.

and one two weeks after operation; all died from contralateral involvement. There were no deaths due to the operation as such.

Four, perhaps five, are "cured," using the word in Sauerbruch's sense of having returned to work, being free of cough and sputum, or with sputum

consistently free of tubercle bacilli. Five are much improved: some of them are recent patients and may attain a "cure." Three are somewhat improved: their future is questionable. The longest period of observation is four years.

There were five patients with phrenectomies: two operated on as a test operation are dead; three others, in whom the operation was followed later by thoracoplasty, are doing well.

There were four intrapleural pneumolyses after incomplete artificial pneumothorax. One patient is cured; three are much improved. Two of these operations were followed by empyemas which for a time frustrated the result, but these were overcome in the end.

Various types of thoracoplasty were performed on cadaveric chests. The chests were filled with plaster-of-Paris; frontal and transverse sections were made of the casts (Figs. 1-3).

Dr. Edward S. Wells, Saranac Lake, N. Y.: Dr. Archibald has said quite a little about the fact that the cavity cases don't do so well, and about avoiding apicolysis in case of a large apical cavity. Dr. Meyer, in discussing indications for the operation, has mentioned fibrous cases with cavities as probably the best type in which to operate. In the few I have done, it has seemed to me that the patients with the cavities did the worst of any, that is, the larger the cavity the worse the patient does, and the less satisfactory the result. Out of sixteen or eighteen that I have had, the two or three that have had large cavities have turned out the worst of any; not that the patients died, but the cavities are not obliterated; as yet I haven't seen any cavity obliterated by the operation. The cavity starts as a large, round hole that one sees in the roentgenogram, and the patient has a lot of sputum. After the operation has taken place, the cavity is reduced, and, after several months, the cavity comes down more and more, but when it seems to reach a point at the end of nine months or a year when the cavity that was formerly as big as an egg is down to the size of a walnut, and there it stays and doesn't seem to change from one roentgen-ray examination to the next; the patient at this time raises a consistent amount of sputum every day and is dissatisfied. Some of these patients come to me and complain that the operation was supposed to cure them of their cough and stop their sputum and now, a year after the operation, they are still raising a certain amount, not as much as before, perhaps, but quite a lot. I don't know what to do for them.

It seems to me that one of the problems we have yet to work out is the treatment of these cavities. I had one patient that I didn't want to operate on. He begged for it and said that he was ready to die, and that if there was any chance at all of helping him, to do it; so we accepted him. It turned out better than anybody expected and inside of three months the man gained between 45 and 50 pounds (20 and 22 kg.). He had been emaciated from vomiting nearly everything he ate, on account of his hard cough, and he began at once to keep down his food and to improve in every way. Then he settled into a sort of chronic state with this cavity trouble that I am speaking of, and I later did an apicolysis trying to collapse a cavity in his apex. I did it according to Dr. Archibald's technic, working in front and putting the pectoral muscles in as a pad. That didn't do the patient any harm, but it didn't accomplish anything, and later, about two and a half years after the first operation, I operated on him again in the back. The cavity was right along the spine, under the ribs in the back, and was a sausage shaped hole about 5 inches (12.7 cm.) long. I did what amounted to an apicolysis in the

back instead of in the front, got to the cavity from the under side of the ribs, and tried to push the walls of it together. The shock was too great and the man's heart gave out; he died two days later.

This secondary posterior operation was about the most difficult task I have ever undertaken, as the periosteum left at the first operation had regenerated new bone, strips of which had fused together in all directions.

I have done only two phrenic operations, but in the second one I used a transverse incision instead of a longitudinal one, back of the sternomastoid, and got a better exposure of the nerve than I did with the up and down one, and was able to work more easily all the way through. This left the man without any visible scar, which I think is quite an advantage, especially in a woman.

Dr. Leonard Freeman, Denver: Some fifteen years ago, in the early days of extrapleural thoracoplasty, I devised a method of treating certain cases in which an apical cavity existed. This depended on taking out two or three ribs and then applying pressure by an ordinary spring truss, such as is used for a hernia, with one pad on the back, the spring passing over the shoulder, and another pad in front, over the area from which I had taken the ribs. One of these ordinary red rubber sponges, such as may be bought in a drug store, makes a suitable pad. The compression of the spring seems to be about right. I have succeeded in at least improving and, as far as I know, in curing several cases in which large cavities existed this way.

Dr. Archibald speaks of the "collapse" of the lung in the Sauerbruch operations. It is true that the word "collapse" is a proper one to use if only a partial operation is done, but if a complete Sauerbruch operation is done, "compression" is a better word to use. I have heard Sauerbruch himself very carefully explain that when the complete operation is done, the lung is not collapsed; it doesn't collapse of itself or by the effect of atmospheric pressure, but the whole thorax rotates, as I think can be seen in the specimen which you have here. It is this rotation of the ribs that causes compression of the lung, and in doing these operations if one takes out a sufficient number of ribs, rotation immediately takes place, and, in a short time, the gap is almost closed.

Dr. Edward W. Archibald, Montreal: I am glad to hear from Dr. Meyer that, according to Brauer, phrenicotomy should be done last, if at all, and not as a preliminary, on account of its practically equalling a dose of tuberculin. I have preferred not to undertake that procedure unless I knew exactly what it was going to do, and I have been waiting for the experience of others.

Dr. Hedblom's tentative proposal to use his graded thoracoplasty procedure in the treatment of pulmonary tuberculosis impresses me very much in this sense, that it would, as he does it, do away with the danger of the original Brauer-Friedrich operation, which, nevertheless, as we all recognize, gives the largest degree of compression or rather collapse of the lung. If you take away the ribs in their entirety, it must be a collapse and can hardly be a compression. In any case, we are inclined to admit, and Sauerbruch admits, that if you take away the ribs in their whole length, you are more apt to get a "practical cure." On the other hand, if you do it subperiosteally, by the time you have come to the third or fourth stage, or even, sometimes, the second stage, leaving more than two or three weeks between, there occurs a very large reformation of new bone. My objection, in a general way, to the three stage operation and to leaving too long a period between the first and second stages lies in that fact that new bone does form, and so, when one comes to do the second or the third stage, one is thereby prevented from getting as great a collapse

as when the operation is completed in one stage. In that sense, Dr. Eloesser is perfectly correct; I think if one can do it in one stage without danger, it is better for lung compression. As a routine, because of its danger, I am not willing to do the one stage operation. If Hedblom's graded thoracoplasty should prove, in spite of this formation of new bone, to give as good a compression or collapse as one can get otherwise, I should be inclined to adopt it for cases in which the ordinary two stage operation has proved insufficient.

Dr. Butler discussed the resection of the clavicle. I thought that Sauerbruch and others had abandoned this procedure. I am not prepared to say that they were right in doing so, but I have never felt that it was really necessary. It is perhaps indicated in the case of large apical cavities, but I doubt the possibility by mere compression of obliterating completely such large cavities. The slight extra compression obtained by adding resection of the clavicle is hardly sufficient to justify the greater deformity. I have, for instance, found cures to occur even in cases in which I did not resect the first rib.

Dr. Eloesser has contributed something of value in demonstrating the necessity of the first rib resection; but I would point out that in a great many cases the apex is thoroughly fibrous and stiff, and when one takes out the first rib one gets absolutely no insinking of the lung at that point. If the apex does sink in, undoubtedly the rib resection helps; but, frequently, the addition of the first rib resection has seemed to me to add nothing of value to the procedure. Nevertheless, I have adopted it as a routine because I cannot be sure but that its resection will do good. Dr. Wells' cases were extremely interesting, and myself I feel that in the case such as he described in which there is a large apical cavity which has not been obliterated by the posterior operation and by a subsequent apicolysis, it must be useless to try and obliterate it by posterior apicolysis. In such cases, I propose, in the future, to open the cavities widely, partly according to Gekler's method (also done by Sauerbruch), but possibly also to add Evarts Graham's plan of massive cauterization. I realize that that method can never accomplish the same results in tuberculosis as in bronchiectasis, because there is infected tuberculous tissue right down to the root, and we can never burn away all tuberculous tissue sufficiently deep into the hilum, because of the danger of bleeding. Consequently, there would remain a large open tuberculous wound with bronchial fistula. Still, I think we must look forward to using it in certain cases.

CARCINOMA OF THE THORACIC PORTION OF THE ESOPHAGUS

REPORT OF A CASE IN WHICH OPERATION WAS DONE ELEVEN YEARS AGO

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NEW YORK

In June, 1913, I published in *Surgery, Gynecology and Obstetrics* an article under the title, "The First Successful Case of Resection of the Thoracic Portion of the Esophagus for Carcinoma." Since then, and particularly in the last few years, inquiries as to the final outcome of the case have reached me so frequently that it appears there exists a desire for a follow-up report on that case.

The method of operating employed in the case involved a new principle in the mode of surgical attack on the new growth, namely, the exenteration of the esophagus from the thoracic cavity. I was led to devise this method because a study of previous cases, in which operation was done by other surgeons who had employed various methods of intrathoracic resection and anastomosis, seemed to prove to me that it would be useless to make any further attempt with the then existing methods, as they had been tried out by some of the greatest masters and had invariably resulted in failure.

Besides this new principle, the operation also brought out a few technical details, the most important of which probably was the mode of access, consisting in an incision in the seventh intercostal space almost in its entire extent and a continuation of that incision upward on the back by cutting through the seventh, sixth, fifth and fourth ribs at some point between their angles and their tubercles. In an article,[1] I called that incision the "normal thoracic incision" for cases of thoracic surgery that required a thorough exposure of the pleural cavity.

Now, as to the case in question.

Mrs. H. T., aged 67, had been suffering from dysphagia for nine months before the time when I performed a gastrostomy on her, being unable to swallow anything but fluids for the last six weeks. The carcinoma extended from behind the lower border of the arch of the aorta downward for nearly 2 inches (5 cm.). Figure 1 shows the outlines of the esophagus. The resection of the esophagus was performed, March 14, 1913, at the German Hospital, New York City, now known as the Lenox Hill Hospital. The patient was placed on her right side, with the left arm up and well forward. The "normal

1. Torek, Franz: The Operative Treatment of Carcinoma of the Oesophagus, Ann. Surg., April, 1915.

Fig. 1.—Outlines of esophagus; site of tumor may be recognized by constriction.

Fig. 2.—Twelve days after excision of esophagus; dark line, a nitrate of silver mark made the day before operation, indicates the incision which runs through the seventh intercostal space and posteriorly continues up to the third intercostal space. The anterior end of this incision is seen in Figure 7.

thoracic incision," mentioned in the foregoing, was made (Fig. 2), i. e., an incision down to, but not through the pleura in the seventh intercostal space, from the posterior end of which it was extended upward to the third intercostal space, the latter, vertical portion of the incision exposing the fourth, fifth, sixth and seventh ribs between the angles and tubercles. This part of the operation was performed under local anesthesia. While the blood vessels were tied, general anesthesia was induced. Then the larynx was intubated for intratracheal insufflation anesthesia. The pleura was then opened by incision in the seventh intercostal space, and the seventh, sixth, fifth and fourth ribs

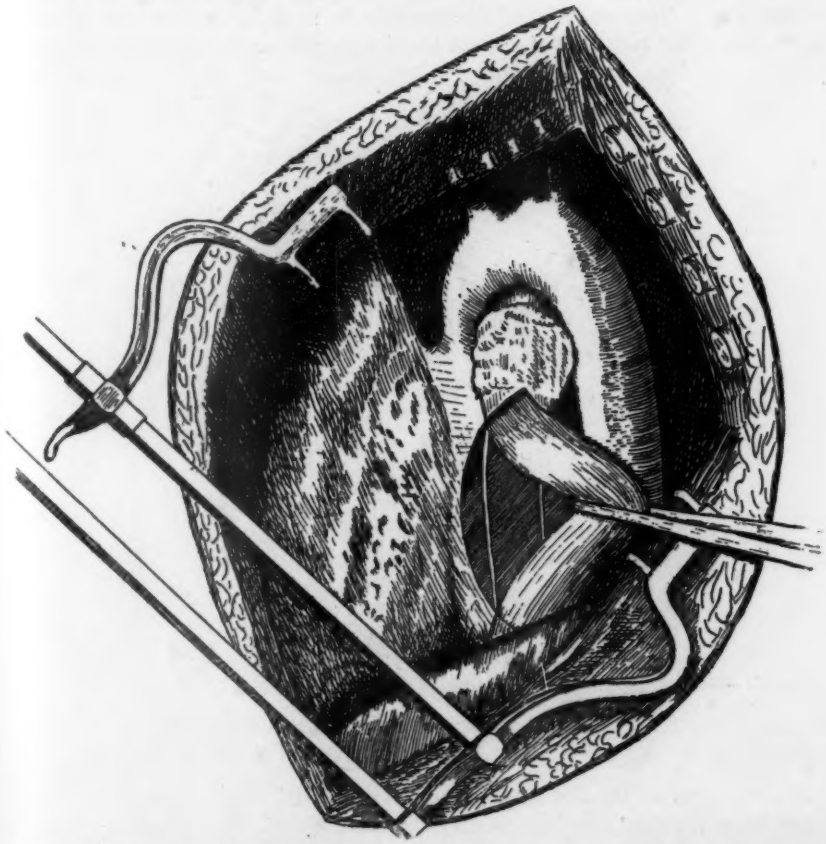

Fig. 3.—First step in dissecting out the esophagus; the lung has been displaced forward; the mediastinal pleura has been incised over the uninvolved lower part of the esophagus which has been lifted out of its bed and is held by a tape; the two vagi have been detached. The tumor is seen below the arch of the aorta.

were divided posteriorly, the intercostal vessels being secured. A Balfour abdominal retractor was inserted to serve as rib spreader. Extensive adhesions between the lung and the parietal pleura were present, the greater part of the lower lobe being attached both to the costal and the diaphragmatic pleura. The firmest adhesions were encountered at the apex. All these were separated.

The tumor was found in the position mentioned in the above and was fairly fixed. The partly collapsed lung was now brought over to the front and the mediastinal pleura inspected. The uninvolved part of the esophagus below the tumor was to be dissected out first. We knew it lay between the aorta, which occupies the posterior part of the mediastinum, and the heart, which is in the anterior mediastinum, and there the pleura was incised, exposing the esophagus. This part of the esophagus was separated from the connective tissue surrounding it and from the vagi and was lifted out of its bed (Fig. 3), after which the dissection was continued upward. At the site of the tumor, dissection was much more difficult, necessitating division of some of the branches of the vagi. This occasioned no appreciable modification or change in the character of the pulse. The dissection of that portion of the esophagus which

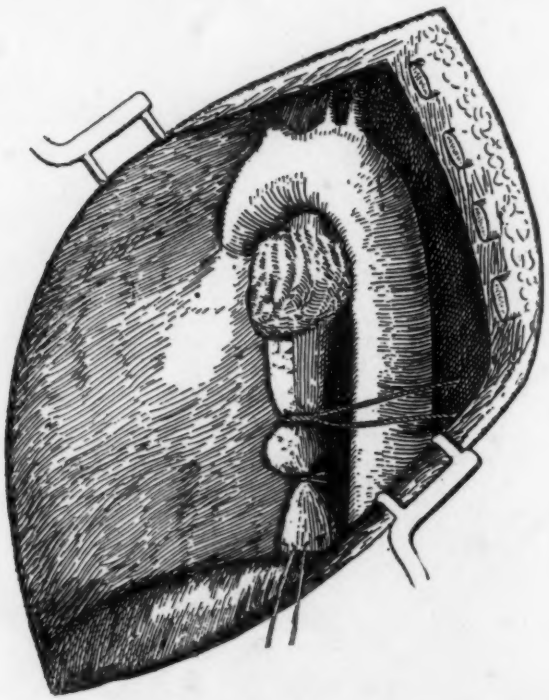

Fig. 4.—Double ligation of esophagus and insertion of purse string suture below lower ligation previous to division of esophagus.

passed behind the arch of the aorta presented great difficulties, which were finally overcome by dislodging the aorta at that site and lifting it forward after having ligated and divided a number of its thoracic branches. The tumor was attached to the left bronchus which sustained a longitudinal cut during the progress of the separation of the tumor from it. The cut in the bronchus was, later in the course of operation, sutured with silk. Next, the esophagus was loosened from its attachments its entire length up to the neck, and there, under the guidance of a finger introduced intrathoracically up to the neck, an incision into the skin at the anterior border of the left sternocleidomastoid muscle was made with the other hand from without. Through this incision, the esophagus was to be drawn out.

The liberation of the esophagus was now complete except for about an inch (2.5 cm.) above the diaphragm. Before the esophagus was divided, surrounding parts were protected by gauze pads, and a double ligation of the organ was applied at a safe distance below the tumor. The upper of these ligatures was of strong silk, and the ends were left long, so that these could be used for traction when the esophagus was brought out through the neck. The lower ligature was of finer silk, to facilitate its proposed invagination. At this site, the esophagus was also crushed with the aid of a Payr's duodenum crusher. The purse string suture, to be used for invaginating the lower stump, was also inserted at a distance below the lower ligature before the esophagus was cut (Fig. 4).

The esophagus was divided by the aid of a cautery between the two ligatures, the upper stump being cauterized with particular care, the lower stump

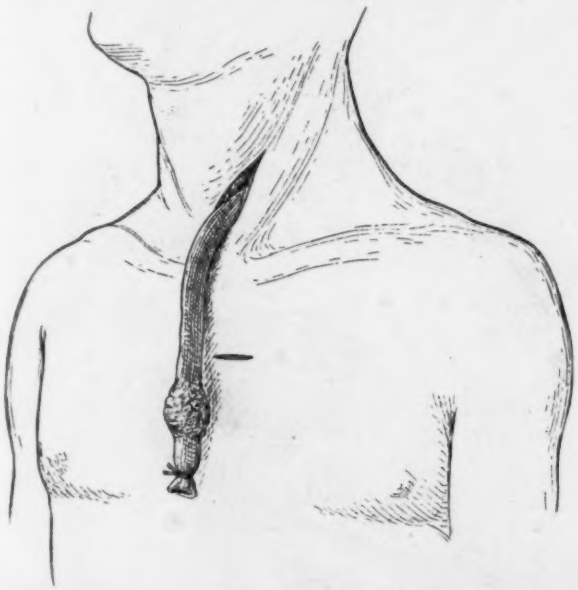

Fig. 5.—Esophagus eviscerated through incision in neck; second incision at front of chest, transverse in direction. These two incisions represent the ends of a subcutaneous tunnel for reception of the esophagus.

was invaginated and the esophagus brought out through the incision in the neck (Fig. 5). A second purse string suture was placed to invaginate still further the stump, and the thorax was closed, silk threads being used to hold the seventh and eighth ribs together. No drainage was employed.

The esophagus, which was hanging out from the wound in the neck, was to be placed under the skin of the chest. For this purpose, a transverse incision in the skin was made corresponding to the site where the carcinoma was to be amputated (Fig. 5). This site corresponded to the second intercostal space. Between this new incision and the one in the neck, the skin was undermined and the esophagus drawn through. The carcinoma was now cut off, and the end of the esophagus was sutured to the skin. The neck wound was closed.

Fig. 6.—Specimen cut open to show mucous surface.

Fig. 7.—Twelve days after excision of esophagus, free end of gastrostomy tube introduced into esophagus for purpose of feeding. Incision in neck (Fig. 5) has healed and is scarcely recognizable.

The pathologic examination proved the new growth to be a squamous cell epithelioma. It was a ragged, indurated ulcer, measuring 4.3 cm. from above down and extending over the entire circumference of the mucosa excepting a small strip about 8 mm. wide (Fig. 6). In one region, it had grown through the external coat, presenting itself in the shape of a nodule about 1 cm. in diameter.

The recovery was uneventful. Pulse, respiration and temperature had become normal on the fifth day. The highest temperature was 102 F., on the third day after operation. The stitches were removed, some on the fourth day, the remaining ones on the seventh day, when the wound was completely closed.

Fig. 8.—Patient, May 18, 1924, eleven years two months after operation.

On the eighth day, the free end of the gastrostomy tube was introduced into the cut end of the esophagus at feeding times (Fig. 7), and, from then on, the patient nourished herself again by swallowing her food.

Now, more than eleven years have passed, a sufficiently long time to test whether the case turned out successfully, not only as to cure but also as to comfort and happiness. The patient, who is now 78 years old, has practically no trouble with the feeding problem. Anything that can be cut or chewed into a finely divided state will readily pass through the tube into the stomach. She is cheerful and happy, takes great interest in the condition of house and garden and everything that goes on around her, and is enjoying very good health. A photograph taken, May 18, 1924 (Fig. 8), eleven years and two

months after the operation, showing the tube in position, if compared with the early picture, will convince one that those eleven years of eating with a rubber esophagus have had no deleterious effect on her well-being and appearance; in fact, she appears to have improved in both.

There are enrolled in the membership of this association two other surgeons who have successfully operated in cases of carcinoma of the thoracic esophagus: Lilienthal by the extrapleural route, and Eggers by my method.[2] Although Lilienthal's patient has since died of a recurrence, the possibility of success was certainly demonstrated, and the recurrence, in his case, was doubtless largely due to the fact that, owing to misinterpretation of the roentgenograms, his exposure of the tumor was not as accurate as it otherwise would have been, and, in consequence, the resection did not go as wide of the affected part as it should have. In operating according to my method, this particular error is, of course, impossible, as the entire thoracic esophagus is exposed.[3]

The main object of this follow-up report is to clear up some misconceptions regarding the condition of a patient subsequent to operation. The fact that my patient has led a comfortable and happy existence for more than eleven years should go far toward counteracting the standpoint of those physicians who still hold the preformed opinion that a patient with carcinoma of the esophagus is better dead than operated on. This pessimistic point of view is very harmful; for the patient, in consequence, is treated expectantly until he becomes in time half starved, his power of resistance is reduced to a minimum, and his tumor has grown to such an extent that its removal becomes anatomically impossible. It is therefore imperative for the profession to understand that the patient's salvation depends on an early operation.

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2. We should mention also the case of Dr. Carl A. Hedblom, which, although it was a case of carcinoma of the cardia, was attacked by the transthoracic route. His method was similar to that of Zaaijer, published in 1913.

3. Altogether there were operated on by my method at our hospital twenty-six patients, with two recoveries. In this series, we have made many errors in operating in cases that were beyond the possibility of successful surgical removal, so that the small percentage of recoveries is ascribable to over-enthusiasm and inexperience, mistakes that would, to a reasonable extent, be cut down in the future.

RESECTION OF THE THORACIC PORTION OF THE ESOPHAGUS FOR CARCINOMA

REPORT OF A SUCCESSFUL CASE

CARL EGGERS, M.D.

NEW YORK

The mortality in cancer of the esophagus is 100 per cent. if left untreated. It is also 100 per cent. with conservative measures and with the treatment by radium and roentgen ray. Surgery, on the other hand, has a few successful cases to its credit, and it would therefore seem that in any given case the decision would be in favor of operating. With the high mortality of the present time, however, many physicians decide in favor of inactivity, of doing nothing, or at most a gastrostomy, in order to have the patient live as long as possible. This attitude of pessimism is shared by many surgeons, who have become discouraged in the face of repeated failures. In certain cases, this attitude is no doubt justifiable, cases in which the prolongation of life is of great value, even though it be but a few months, and in which it is felt that the risk connected with an operation is too great.

On the other hand, if we consider that one of the reasons for the poor surgical results is the fact that the patients are often referred to the surgeon only when everything else has been tried, when they have become weakened, emaciated and discouraged, and they themselves beg for relief, it seems proper to plead with the medical men to send their cases to the surgeon earlier. It is recognized, however, that this is not always easy, that the diagnosis is connected with difficulties, and that by the time the symptoms become fully developed, the disease has sometimes progressed so far as to make extirpation impossible.

In order to get better results therefore, the first essential is to recognize cases early. Difficulty with swallowing and high epigastric or substernal pain should always arouse suspicion and call for an esophageal examination. Physical examination does not help. Passing a bougie often aids in the diagnosis, although that also is often negative in an early case. We have to rely on the roentgen-ray examination, therefore, to make the diagnosis. This should be followed by an esophagoscopy, perhaps combined with a biopsy. If all these measures fail, and the symptoms persist, the patient should remain under observation until a diagnosis can be made or the condition definitely ruled out. At times, an exploratory thoracotomy is indicated, because the operation of extirpation of a carcinoma of the esophagus is so serious that it should not be attempted without a positive diagnosis.

If a carcinoma of the esophagus is left untreated for a while, it will gradually involve all the coats, become adherent to the arch of the aorta, a bronchus, lung or the opposite pleura. The lymph nodes may become involved, the vagus nerves incorporated in the tumor and altogether the growth may be in such a state of advance that its removal is not possible. Such cases have to be excluded in the very beginning. Under consideration for extirpation can come only such patients as are operable, in whom the tumor can be removed without seriously damaging surrounding structures.

With an established diagnosis of carcinoma of the esophagus, the important thing to do, therefore, is to determine its operability. This cannot be done by physical examination alone, nor by the addition of roentgen-ray study, although the latter may indicate by the position and extent of the shadow that the lesion can probably not be removed. A more important aid is the exploration of the cardiac region of the stomach and the liver at the time the gastrostomy is being done. Abdominal metastases would naturally not encourage one to attempt to remove the primary lesion. But even with all these aids, it is sometimes not possible to determine the operability until the tumor is actually exposed and attempts are made to remove it. Whether, in the face of a penetrating tumor and extensive adhesions, it is advisable to continue the operation, will naturally depend on the judgment of the individual surgeon. That one must not be discouraged too readily is evidenced by Torek's case in which the tumor was adherent to the left bronchus and in which the latter was cut and later sutured without untoward symptoms. In my own case, also, the right pleura and the vagus nerves were firmly adherent to the growth, but caused no serious difficulty.

Given a patient with an operable carcinoma of the thoracic portion of the esophagus, what is the best method of procedure? In order to answer this question, it is necessary to know the various causes most likely producing death or leading to complications, and then to adopt some plan which will avoid or counteract them. From a study of the literature and from personal experience, we know that shock, acute pneumothorax, injury of the vagus nerves, pneumonia, traumatic empyema and leakage from the stump or suture line are the various causes producing death. Many and ingenious are the methods devised to avoid or overcome them. A number of excellent articles have appeared in the literature dealing with this subject. I desire particularly to call attention to the one by Fischer,[1] presented before the American Association for Thoracic Surgery, in 1922, in which he gives a review

1. Fischer, Hermann: Surgical Treatment of the Esophagus, Arch. Surg. **6**:256 (Jan.) 1923.

of this subject and a complete bibliography. Torek[2] succeeded in developing a method which meets the demands more perfectly than any other, and by means of it he was able to carry out the first successful operation for the resection of a carcinoma in the thoracic portion of the esophagus. With his incision he gets excellent exposure, which makes it possible to attack the tumor and separate the surrounding tissue with the least amount of damage. The pleura may be pushed off under guidance of the eye, the vagus nerves dissected off or sharply divided and bleeding controlled without difficulty. It is also more easily possible to avoid soiling the field. Thus, two of the causes of death mentioned in the above, injury of the vagi and a traumatic empyema due to the soiling of the field at the time of operation, may be guarded against. Leakage from the stump is avoided by bringing the entire upper end of the esophagus outside the thorax and transplanting it under the skin.

The other possible causes of death, shock, acute pneumothorax and pneumonia, have to be considered in each case individually. In a case of this character, shock will always play an important rôle, because the operation is a formidable one. It is therefore important to put the patient in as good physical condition as possible: (1) by establishing a gastrostomy which will allow administering food in large quantities; (2) by keeping the mouth clean and removing any possible foci of infection, and (3) by attention to the heart and kidneys. Unfortunately, many of these patients are in advanced years, men with a bad heart, high blood pressure and emphysema. At the time of operation, shock can be guarded against by keeping the room warm, avoiding loss of blood, not handling the tissues roughly, especially avoiding pulling or pressing on the heart and aorta, and by inflation of the lungs at intervals. The latter procedure can readily be done by the anesthetist. If the vagus nerves cannot be separated from the tumor, but have to be sacrificed, one should divide them sharply just as low as possible.

Acute pneumothorax is guarded against by appropriate measures. Inflation of the lungs at intervals during the operation is of great value. At the end of the operation, the lungs should be inflated, so that all air is forced out of the pleural cavity. In order to be certain of this, the intrapharyngeal insufflation may be continued until the dressings are in place and the patient returned to bed. In case drainage is used, the air may be forced out of the pleural cavity while the end of the tube is under a fluid level in the drainage bottle.

It may be assumed that postoperative pneumonia is more likely to develop in these cases because of the long exposure of the lung, its

2. Torek, Franz: The First Successful Case of Resection of the Thoracic Portion of the Esophagus for Carcinoma, Surg., Gynec. & Obst., June, 1913, pp. 614-617.

handling and compression. It can best be avoided by improving the patient's circulation before operation and by inflating the lungs from time to time during the operation. Aspiration pneumonia should not occur more often than ordinarily. Mouth hygiene before operation is essential. The esophagus should be empty before the operation is started.

Since Torek's [2] first successful resection in 1913, only one other patient has survived the operation long enough to be called surgically successful. This is Lilienthal's [3] case, in which operation was done, in 1921, by the extrapleural method, and the patient lived for several months, when he died of a recurrence. Although many patients have been admitted to the Lenox Hill Hospital with the diagnosis of carcinoma of the esophagus since 1913, the large majority proved to be inoperable. In only a small percentage of cases was removal actually attempted or carried to conclusion, and in most of these the outlook from the beginning was unfavorable, either owing to a poor general condition which could not be improved, or to an advanced local condition which often made complete removal impossible even after it had been attempted. But, although there have been no additional cures, much has been learned, and a number of patients have survived the operation several days, dying of a complication not always directly connected with the esophagus. Much pioneer work has had to be done, and a surgeon had to be unusually optimistic to continue in the face of difficulties and adversities. It has been my good fortune to be associated with such ardent workers as Dr. Willy Meyer and Dr. Franz Torek, and to profit by their enthusiasm, tenacity and knowledge. There have been many problems to solve, but there is reason to believe now that a greater degree of hope can be held out to these unfortunates than was formerly possible.

Torek's patient is still living, eleven years after operation, and I have recently been so fortunate as to operate successfully on a patient with a carcinoma of the esophagus just below the arch of the aorta. The same general technic advised by Torek was followed, differing from it only in several details.

The history of this case is as follows:

REPORT OF CASE

J. P., aged 38, admitted to Lenox Hill Hospital, service of Dr. Jacob Kaufmann, March 11, 1924, had as his chief complaints dysphagia, vomiting, loss of weight and girdle pain in the upper abdomen. They had been present only six weeks, beginning with difficulty in swallowing, followed by a sensation of

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3. Lilienthal, Howard: Carcinoma of Thoracic Esophagus, Extrapleural Resection and Plastic, Ann. Surg. **74**:259 (Sept.) 1921.

pressure in the upper part of the abdomen, as if he were encircled by a tight belt. At times, the pain was rather sharp, lasting a few minutes and then disappearing. After about two weeks, the pain came on only after eating. At the time of admission, he would induce vomiting in order to relieve his pain. Swallowing had become progressively more difficult, and for the last three weeks he had been unable to take anything but liquids, and even they had to be taken slowly and in small quantities, as too large a quantity would at once cause pressure and pain, leading to induced vomiting. He had lost 25 pounds (11.3 kg.) in weight since onset, and had become very constipated.

The past and family history had no bearing on his present trouble. Some years previously, he had been a rather heavy drinker. He smoked from three to five cigars daily.

Fig. 1.—*A*, esophagus after administration of bismuth meal showing almost complete stricture in lower thoracic portion; *B*, three days after administration of full doses of atropin.

The physical examination was negative; his weight was 129 pounds (58.5 kg.). An esophageal bougie met an impassable stricture 15 inches (37.5 cm.) from the teeth. The Wassermann reaction was 2+. The history was negative. Roentgen-ray examination of the esophagus, by Dr. William H. Stewart, showed a deformity about 3 cm. above the diaphragm, with almost complete obstruction, which was interpreted as a carcinoma. The examination was repeated after three days administration of atropin, and showed no change (Fig. 1).

March 23, the patient was transferred to the surgical department. Since admission, his weight had steadily gone down, until it reached 114 pounds (51.7 kg.), a loss of 15 pounds (6.8 kg.) in less than two weeks. The most urgent indication was to feed the patient. A Witzel gastrostomy was therefore

done, under local anesthesia, March 24. Exploration of the upper part of the abdomen at the same time showed no liver metastases and no glands below the diaphragm, and palpation through the diaphragm failed to reveal a tumor. Owing to the youth of the patient, other diagnoses than carcinoma were considered. For this reason, the Wassermann test was repeated, and this time reported as 3+, which induced us to start active antisyphilitic treatment. A week after the gastrostomy, an esophagoscopy and a biopsy were done, by Dr. John D. Kernan, which substantiated the diagnosis of a tumor in the lower part of the esophagus. The pathologic examination was done by Dr. Fred D. Bullock. It showed a squamous cell epithelioma. The patient was put on a well balanced mixed diet, but in spite of that he gained only slowly, so that it became necessary to postpone the radical operation longer than had been

Fig. 2.—Posterior and lateral aspect, showing extent of thoracic incision, which healed by primary union; stab wound used for drainage.

desired. After about a month, he had gained 5 pounds (2.3 kg.); he felt stronger, and offered a chance to stand an extensive operation.

April 24, the operation was performed. An incision was made along the entire left seventh intercostal space, with the patient lying on the right side. The posterior end of the incision was then carried upward over the seventh, sixth, fifth and fourth ribs (Fig. 2). The ribs were divided near their tubercles and, by means of a rib spreader, the thorax was opened wide. There were no adhesions, and no metastases were noted. The esophagus was easily exposed below the arch of the aorta, liberated and lifted out of its bed by a tape passed underneath it. It was followed downward until the tumor was reached. The latter was found to be situated just above the diaphragm, and to extend upward for from 1½ to 2 inches. It was very hard and nodular, all the coats were involved, and there were dense adhesions, especially with the right pleura.

The vagus fibers were incorporated in the tumor to such a degree that they could not be separated, but had to be sacrificed. They were sharply divided. No disturbance in the patient's condition was noticed. By traction on the tumor it was possible to separate the diaphragm from the esophagus and to bring into view the subdiaphragmatic portion. This part was rather short and made it difficult to place double ligatures and still have enough of a stump for invagination into the stomach. The upper ligature was therefore placed rather close to the tumor, and the esophagus then divided by means of an electric cautery. The lower stump was then invaginated into the stomach by one silk purse string suture. It dropped back beneath the diaphragm at once. A few chromic gut sutures were then placed to close the hole in the diaphragm, and by means of plain catgut sutures the pleural margins were approximated for a short distance to close the posterior mediastinum. The esophagus was now liberated behind the arch of the aorta by blunt dissection, all the small vessels that were encountered being doubly ligated. No real difficulty was experienced. The pleura was now incised above the arch, and the esophagus, with the tumor in its lower end, pulled upward from behind and below the arch. While this was being done, the ligature slipped off, but, fortunately, there was no gross leakage, owing to the tight stricture and the thorough cauterization which had been done. The stump was again cauterized, and several sutures were taken through its lowest end. The esophagus was then freed upward as far as the neck.

On completion of this part of the operation, the thorax was temporarily partly closed, towel clips being used for approximation, and the patient was then turned on his back. An incision was made in the neck along the anterior border of the left sternocleidomastoid muscle. The large vessels were retracted outward, and the esophagus easily brought into view. Traction on it brought out the entire organ, with the tumor in its lower end. It was temporarily placed on the thorax, wrapped in moist gauze.

The patient was then again turned on his side, and the chest reopened. By means of a continuous plain catgut suture, the pleural margins were united from the dome of the thorax to the arch of the aorta. This could be done without difficulty. The neck wound was thus shut off from the pleural cavity and entrance of air was avoided. We were now ready for closure. To drain, or not to drain was a question not easy to decide, but, chiefly on account of the slipped ligature, was decided in favor of drainage. It should be closed drainage in order to keep the lung expanded. A stab wound was therefore made in the eleventh interspace posteriorly, and a half inch rubber tube inserted. It projected into the thorax for a distance of 3 inches (7.6 cm.), and had a lateral and an end opening. It was fastened to the chest wall outside by adhesive plaster strapping. The chest wound was now closed by first approximating the seventh and eighth ribs with interrupted chromic gut sutures. The muscle layers were united by chromic gut, and the skin was closed with silk. A dressing was applied, and the patient then turned on his back.

Attention was now directed to the neck wound. The muscles were closed around the esophagus. A subcutaneous tunnel was established extending from the neck wound to the cartilage of the left third rib, and a transverse incision made at its lower end. The esophagus with the tumor was then drawn through this tunnel and out of the lower incision where it was sutured to the skin margin. The projecting portion containing the tumor was removed with the electric cautery. The neck wound was closed with silk, and a dry dressing applied.

My assistants at the operation were Drs. Herbert Willy Meyer, Walter Stenson and Charles Oppenheim. The entire operation had lasted three hours and five minutes. It had been well borne. The anesthesia had been very skilfully given by Dr. George Muehleck, who used gas-oxygen-ether with the Gwathmey apparatus. In the course of the operation, the lung had been frequently inflated; this was accomplished by increasing the pressure. It always produced a marked improvement in the condition. This same practice was resorted to while the thorax was being closed, and at the end of the operation, with the patient in bed, the drainage tube was opened while the lung was being expanded, forcing out all air. The tube was then clamped, put under fluid level in a drainage bottle and again released.

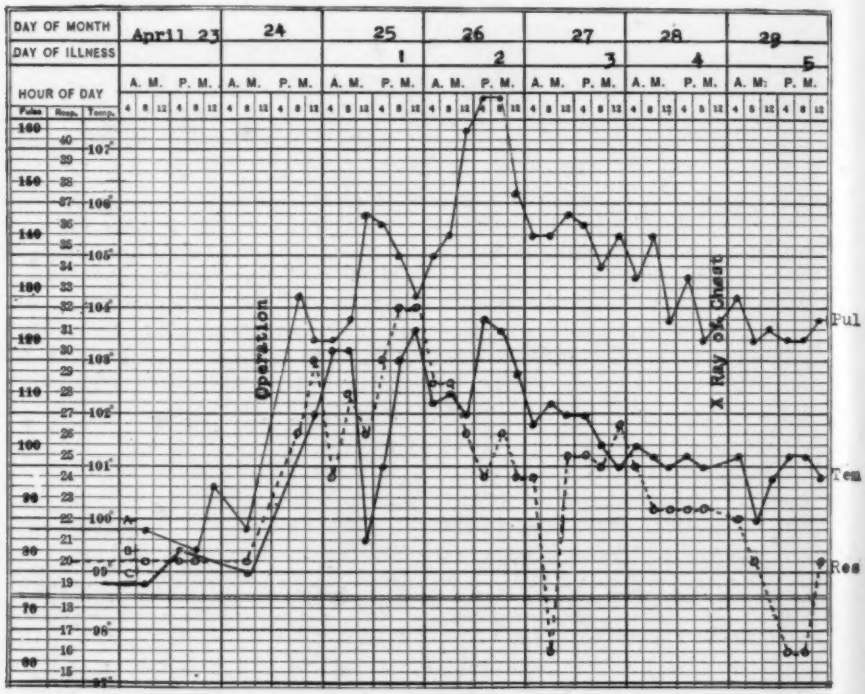

Fig. 3.—Temperature chart first week after operation; immediate sharp rise, reaching its highest point on the evening of the second day, and then subsiding, but continuing about 101, owing to gangrene of esophagus stump; *A*, pulse; *B*, respiration; *C*, temperature.

Two hours after operation, the patient was fully conscious; his pulse was rapid and thready (142) but regular. A hypodermoclysis of 1,000 c.c. Ringer's solution was given into the thighs. The convalescence of the first few days was rather smooth, except toward the end of the second day, when the patient was restless, had an anxious expression, a temperature of 103.8 F. and a pulse of 160. The temperature, however, gradually subsided (Fig. 3), and the general condition improved uninterruptedly. He was quite cheerful and talkative most of the time. During the first two days, fluid was administered entirely by hypodermoclysis and the rectal drip method, for fear of producing leakage of

the stump at the cardiac end. At the end of that time, it was given through the gastrostomy, at first by the drip method, and later in gradually increased fixed quantities at regular intervals.

The history of drainage in this case is of interest. During the first twenty-four hours, there was a discharge of about 200 c.c. of serosanguineous fluid; during the second and third twenty-four hours, about 50 c.c. each day, and then it stopped. At no time was this fluid odorous, nor did it show the characteristics

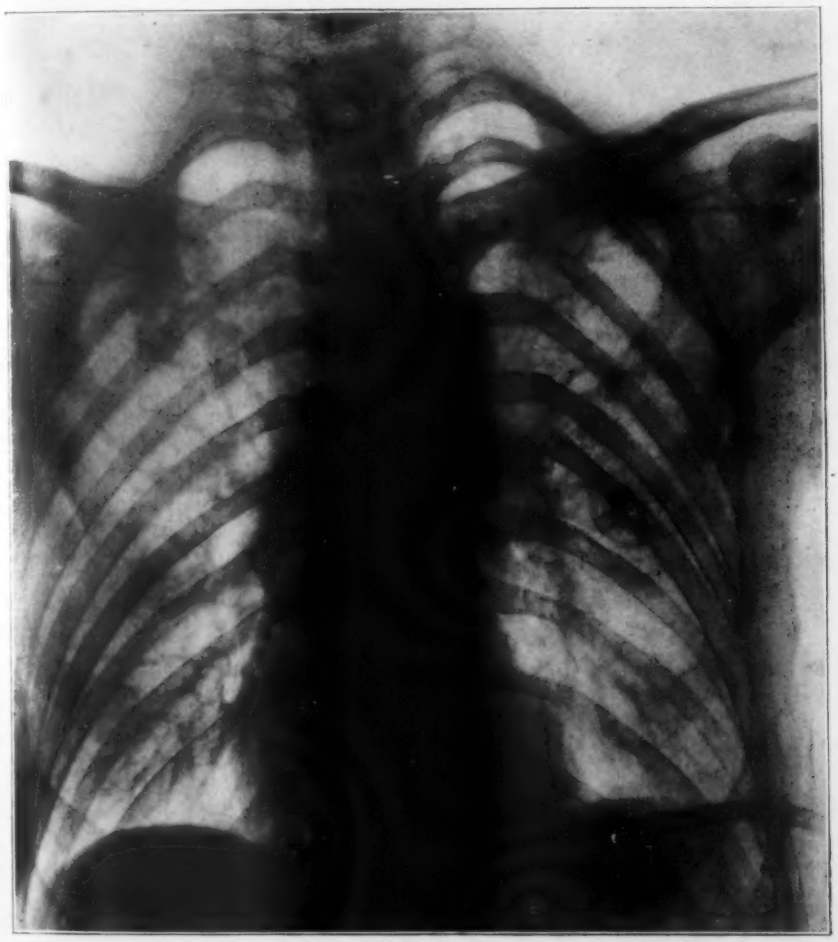

Fig. 4.—Bedside roentgenogram of chest four days postoperative; excellent lung expansion; good apposition of divided fourth, fifth, sixth and seventh ribs, and no fluid.

of pus. Cultures taken from the dripping of the tube, on the third, fourth and fifth days, showed, on the first day, colon bacilli and short chained hemolytic streptococci, and, on the following days, colon bacilli only. During these days, the lung on the operated side seemed well expanded, and breath sounds were audible over the front and back. On the fourth day, a roentgenogram was taken of the chest which showed excellent expansion, no cardiac displacement, good apposition of the divided ribs and no fluid (Fig. 4). The drainage tube

was therefore removed on the following day, after which the stab wound healed without difficulty. The wound in the neck and the long thoracic wound healed by primary union. The sutures of the former were removed on the fourth day, and of the latter on the seventh day. The esophagus stump, on the other hand, did not heal in place quite so smoothly. It was early noticed that there was some gangrene of the lowest end, and the patient continued to have temperature of about 101. On the eighth day, he did not look or feel

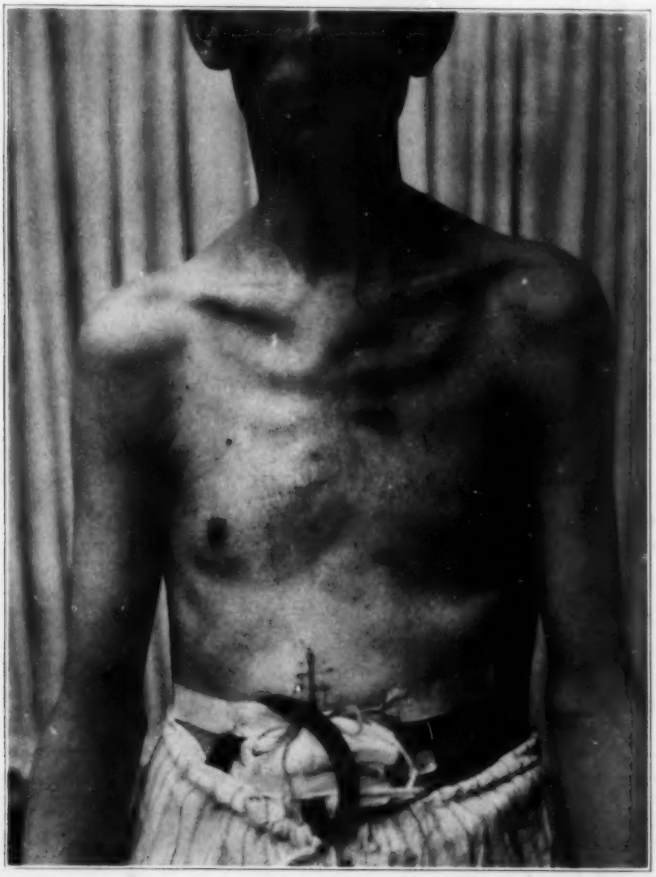

Fig. 5.—Three incisions on the left upper anterior thoracic wall necessitated by the esophagus stump becoming gangrenous; lower two openings have entirely healed, while the upper represents the entrance into the esophagus; gastrostomy tube curled up in the dressing while not in use.

as well as before. There was a conjunctivitis of the right eye, herpes of the lips, and a general appearance of depression and apathy.

Examination showed that the gangrene of the esophagus stump extended up further than that portion projecting beyond the skin. Fluctuation could be elicited next to the buried portion of the esophagus. Under local anesthesia, a 1 inch (2.5 cm.) transverse incision was made about 2 inches (5 cm.) above

the esophageal exit, which was just beneath the sternoclavicular joint. A normal esophagus and no pus were found. Another transverse incision was therefore made further down. A necrotic esophagus and a pus pocket extending upward on the right side were found. The cavity was evacuated, and lightly packed with a Dakin tampon. After this, the patient rapidly improved. The wound became clean, and when the gangrenous portion of the esophagus had been extruded, a good esophageal fistula was established through the uppermost transverse incision (Fig. 5).

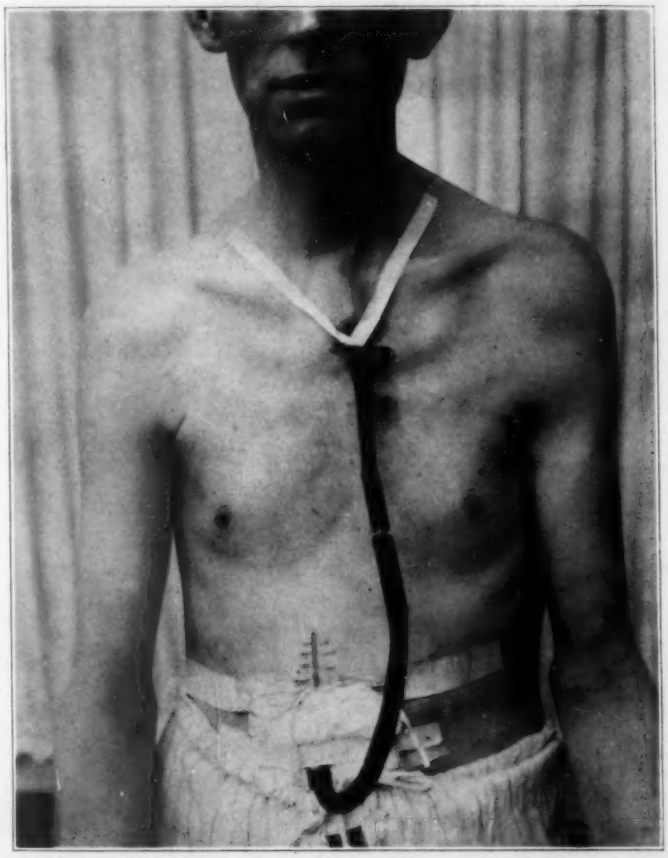

Fig. 6.—Patient with the rubber esophagus, which functions perfectly, in place.

On the fourteenth day, the patient was allowed up in a wheel chair, and daily thereafter. Owing to the presence of a draining wound below the exit of the esophagus, we did not begin giving anything by mouth until three weeks after the operation, and it was four weeks before regular feedings were started. The patient took to this method of feeding eagerly, and, a few days after starting it, he was able to masticate and swallow all articles of food. The mechanism is as follows: A long gastrostomy tube is armed with a rubber cuff about 2 inches (5 cm.) from its upper end, which fits snugly

against the esophageal opening when the tube has been inserted. To insure no leakage during deglutition, the patient makes light pressure with his fingers over the esophagus on either side of the tube. He masticates his food thoroughly, and then swallows it the normal way, the food passing through the pharynx, upper esophagus and then through the rubber esophagus into the stomach. A little more fluid is required than for people with a normal esophagus (Fig. 6). Between meals, the tube may be withdrawn from the esophagus and closed with a cork, or it may be left in place, as the patient desires.

It was considered to be of interest to study the function of the stomach in the presence of a gastrostomy and to compare the findings under the following conditions:

1. With mastication excluded, saliva in the stomach absent, and both vagus nerves probably completely divided.

Fig. 7.—Gross specimen of carcinoma of esophagus; division had been made well beyond the tumor, but, owing to extensive cauterization, it appears that the lesion extends up to the line of division.

2. With mastication reestablished, saliva entering the stomach, and both vagi still in the same condition.

The first tests were therefore made after the operation, while the patient was still being fed through the gastrostomy, and were later repeated, after a connection had been established between the mouth and the stomach by means of the rubber esophagus. Chemical analyses were made before and after a regular test meal, and the motor function of the stomach was studied by means of a bismuth meal in the stomach.

From a practical standpoint, the examinations showed nothing remarkable. There was no free acid in any specimen. The total acidity varied from forty

to sixty. Lactic acid was present in all specimens. The mobility of the stomach was good; there was slight six hour retention, which was probably due to spasm, the result of the presence of the gastrostomy tube. There was no twenty-four hour retention.

The specimen removed at operation in the fresh state was about 3 inches (7.6 cm.) long. A deep ulcer with elevated edges took in the entire lumen and produced a stricture so tight that only a small probe could be inserted. All coats were involved (Fig. 7). The pathologic report was identical with that of the biopsy: squamous cell epithelioma. One gland which had been removed showed no metastases.

In reviewing the case, several points deserve special mention. The youth of the patient is of interest, and, in connection with that, the short history of six weeks' duration. The lesion must naturally have been there considerably longer, but no symptoms appeared until there was actual obstruction. The latter increased so rapidly that, at the first roentgen-ray examination, it was pronounced almost complete. The result was a loss of 15 pounds (6.8 kg.) in weight in less than two weeks. Only the early transfer of this patient to the surgical service made it possible to do anything for him at all.

The question of drainage is also of importance. Opinions on this subject differ considerably. Although, in this particular case, drainage was established, and although organisms were actually found in the secretion, I have a feeling that the drainage could have safely been dispensed with. The amount of secretion was so small it could readily have been absorbed. The real reason for drainage was the fact that the ligature of the stump had slipped, and I have the feeling that, in a subsequent case, if there seems any danger of infection, I shall drain, otherwise not.

The patient's condition has been followed closely since he left the hospital. He is gaining steadily, and has become sufficiently strong to want to return to his former occupation.

FURTHER EXPERIMENTAL RECONSTRUCTION OF THE ESOPHAGUS WITH AUTOGENOUS FASCIA LATA TRANSPLANTS *

DUFF S. ALLEN, M.D.

ST. LOUIS

I. Previously reported experimental reconstruction of the esophagus by autogenous fascia lata transplants.
II. Choice of material for replacing the resected portion of the esophagus.
III. Plan of these later experiments.
IV. Experimental data.
V. Stenosis.
VI. Conclusions.

In a previous communication [1] emphasis was placed on the importance of avoiding infection in the operation for resection of the esophagus. The source of this infection was found to be, in the main, from "leakage of the contents of the esophagus. Whether this leakage takes place at the time of operation or subsequently, the result is the same—infection." Another source of infection was pointed out to be that from "epithelial lined tubes of the viscera or the entire viscus which was to replace the resected portion of the esophagus." Infection along any part of the esophagus usually leads to a fatal mediastinitis.

Because of the fact that fascia lata is sterile and since it will unite to the nonepithelialized outer surface of the wall of the esophagus, it was the material of choice for the repair of defects in the wall of the esophagus.

I wish to tabulate again, briefly, the basic operative procedures which have been devised for esophagoplasty:

1. Cervical esophagoplasty by means of skinplasty (1886).[2]

2. Extrathoracic esophagoplasty by means of skinplasty of neck and anterior throax (1894).[3] (In Dr. Torek's successful case, operation was done by a method similar to this.)

3. Inferior extrathoracic esophagoplasty by use of a portion of the jejunum (1907).[4]

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* From the Washington University Medical School department of surgery.

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2. Hacker, V.: Zur Pharyngo und Osophagoplastik, Zentralbl. f. Chir. **18**:121, 1891.

3. Bircher, H.: Ein Beitrag zur plastischen Bildung eines neuen Oesophagus, Zentralbl. f. Chir. **34**:1479, 1907.

4. Roux: L'oesophago jejuno gastrostomose; nouvelle operation pour rétrécissement infranchissable de l'oesophage, Semaine méd., 1907, p. 37.

4. Inferior extrathoracic esophagoplasty by use of a portion of the jejunum and skinplasty (1904).[5]

5. Inferior extrathoracic esophagoplasty by use of a portion of the transverse colon (1911).[6]

6. Inferior intrathoracic esophagoplasty by use of the stomach (1905).[7]

7. Inferior extrathoracic esophagoplasty by use of a tube of the anterior wall of the stomach (1911).[8]

8. Inferior extrathoracic esophagoplasty by use of a tube of the greater curvature of the stomach (1904, 1912).[9]

9. Inferior extrathoracic esophagoplasty by use of the first horizontal portion of the duodenum (1913).[10]

10. Posterior thoracic esophagoplasty by use of skin flaps (1921).[11]

Reconstruction of half the circumference of the esophagus with fascia has been done by Razzaboni and Neuhof:

1. Cervical esophagoplasty by use of the sheath of the rectus and peritoneum (1917).[12]

2. Cervical esophagoplasty by use of the fascia lata (1917).[13]

In each of these operative procedures for resection of the esophagus, it is necessary to open the lumen of the esophagus at the time of operation; in some of them, unsterile transplants are made into the mediastinum. Mediastinitis is a frequent result. This is a serious complication.

In our previously reported experimental reconstruction of the esophagus, a two stage reconstruction of the esophagus was described. Fascia lata was chosen to replace the resected esophagus. A sleeve of

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5. Wullstein: Ueber antethorakale Oesophago-Jejunostomie und Operationen nach gleichem Prinzip., Deutsch. med. Wchnschr. **31**:734-736, 1904.

6. Kelling, G.: Osophagoplastik mit Hilfe des Quercolon, Zentralbl. f. Chir. **38**:1209, 1911. Vulliet, H.: De l'osophagoplastique et de ses diverses modifications, Semaine méd. **31**:529, 1911.

7. Sauerbruch: Die Anastomose zwischen Magen und Speiseröhre und die Resektion des Brustabschnittes der Speiseröhre, Zentralbl. f. Chir. **32**:81-86, 1905.

8. Hirsch, M.: Plastischer Ersatz des Osophagus aus dem Magen, Zentralbl. f. Chir. **38**:1561, 1911.

9. Beck, C.: Demonstration of a Method of Formation of Aplethoric Esophagus, Illinois M. J. **7**:463, 1905. Jianu, A.: Gastrostomie und Osophagoplastik, Deutsch. Ztschr. f. Chir. **118**:383, 1912.

10. Fink, V.: Ueber plastischen Ersatz der Speiseröhre, Zentralbl. f. Chir. **40**:545-547, 1913.

11. Lilienthal, Howard: Carcinoma of Thoracic Esophagus, Extrapleural Resection and Plastic, Ann. Surg. **74**:259 (Sept.) 1921.

12. Razzaboni, G.: Richerche sperimentali sull' autoplastica esofogea per mezzo del trapianto di lembi libri musculo-aponeurotico peritoneali, Policlinico (sez. chir.) **24**:417 (Oct.) 1917.

13. Neuhof, H.: Fascia Transplantation in Visceral Defects, an Experimental and Clinical Study, Surg., Gynec. & Obst. **24**:383-427 (April) 1917.

fascia lata was implanted and allowed to grow around the esophagus before resection. This was done to prevent leakage of the contents of the esophagus after resection. At a second operation, the sleeve was split and the section of the entire circumference of esophagus contained within it was resected. Nine resections of the cervical esophagus and five resections of the thoracic esophagus were done by this method in dogs. Of these, one (thoracic) had leakage through the split in the sleeve of fascia after resection. All the others were successful.

From this previously reported experimental work, the following conclusions were drawn:

1. Infection along the cervical portion of the esophagus may produce a mediastinitis or a mediastinitis associated with a generalized empyema.
2. This infection is the result of leakage of the contents of the esophagus.
3. Stenosis may or may not be found after repair of defects in the wall of the esophagus with fascia lata transplant.
4. It is possible to reconstruct portions of the entire circumference of the esophagus with fascia lata transplants. The result is a tube lined with squamous epithelium derived from the epithelium which normally lines the esophagus.
5. By the two stage operation, the danger from leakage of the contents of the esophagus and autoinfection is reduced to a minimum.

It occurred to us that if we could remove that portion of the esophagus (presumably carcinomatous) which was enveloped by the sleeve of growing fascia lata without splitting the fascial sleeve at the second operation, all danger of leakage of the contents of the esophagus through this split would be eliminated. This might be done by approaching the resection from the interior of the esophagus—through the esophagoscope—after the fascial sleeve had been allowed to anchor itself around the outside of that portion of the esophagus which was to be resected. Another plan of approach—and one which would permit better inspection than could be had through the esophagoscope—is the one described below.

PLAN OF OPERATION

This plan for resection of the carcinomatous area of esophagus without splitting the enveloping sleeve of growing fascia lata aims to shut off the blood supply to the carcinomatous area after the danger of leakage has been eliminated. It is as follows:

If a sleeve of fascia lata is placed around a section of the esophagus, simple ligation of the esophagus near both ends of the sleeve will cut off all the blood supply to the ligated portion of the esophagus, provided that sufficient time has not elapsed between the time of implantation of the fascia and the ligation of the esophagus for a new circulation to establish itself to the section of esophagus through the fascial transplant.

A carcinomatous section of the esophagus which had been enveloped in this manner might be caused to slough in its entirety and to be swallowed as necrotic tissue.

Sufficient time must elapse between implantation and ligation to allow adhesions to form between the ends of the fascial sleeve and the ends

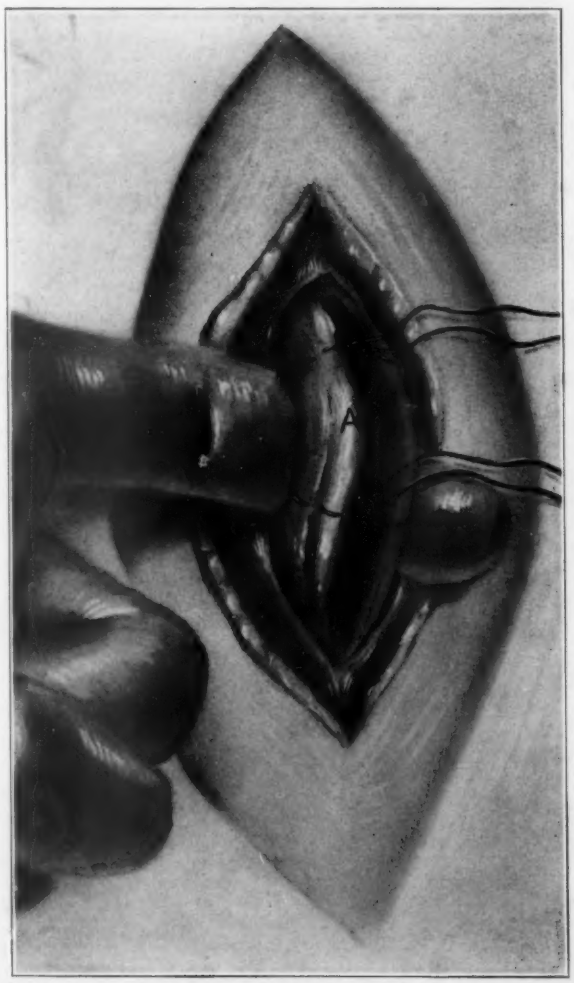

Fig. 1.—First step of first operation; two stout silk ligatures are planted around the esophagus at the ends of the section of esophagus which is to be removed.

of the remaining esophagus to prevent leakage. This can be done experimentally by a two stage operation.

At the first operation, two stout silk ligatures are "planted" around the esophagus (Fig. 1). These ligatures and that portion of the esophagus between them are enveloped by a sleeve of fascia lata (Fig. 2). The

ends of the ligatures are carried out through the fascial sleeve on a needle, but they are not tied (Fig. 3). The lumen of the esophagus is not opened and, experimentally, no leakage of the contents of the esophagus follows.

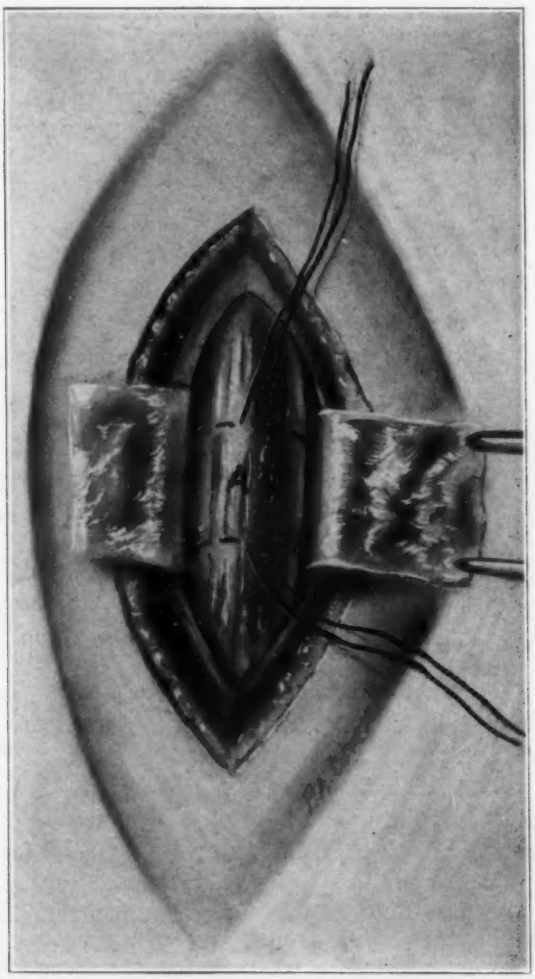

Fig. 2.—The strip of fascia lata is broad enough to cover the section of the esophagus to be removed and also to extend beyond its limits. That portion of the fascia lata which extends beyond the ligature will become adherent to the ends of the esophagus which are to remain after removal of the section of the esophagus, *A*, lying between them. This prevents leakage after sloughing of *A*.

At a second operation, from four to eight days after the first operation, the ends of the ligatures which have been planted around the esophagus are again picked up and tied tightly. The knot is tied inside

the fascial sleeve, the ends are cut short, and the two needle holes in the fascial sleeve are closed by the surrounding connective tissue. The wound is closed. Experimentally, no leakage of the contents of the esophagus follows.

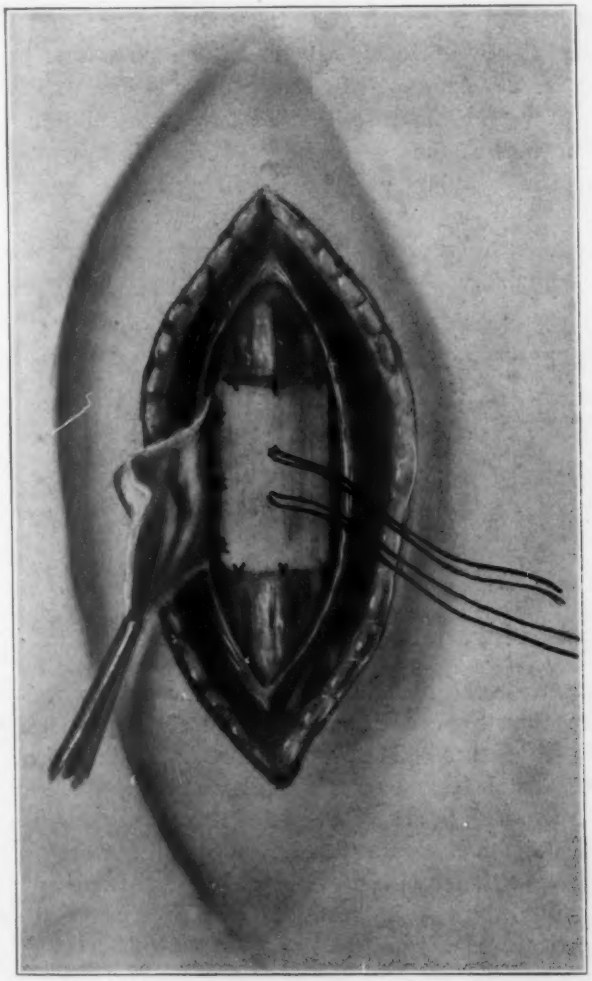

Fig. 3.—The sleeve of fascia lata has been placed completely around the esophagus, ends of the ligatures passing through the sleeve of fascia. These are left long, and are not tied at the first operation. At the second operation, they are tied tightly.

The lumen of the esophagus has not been opened at the first operation, nor is it opened at the second; it opens spontaneously with the sloughing of the section of esophagus which is to be destroyed after the fascial tube has sealed itself.

EXPERIMENTS

Ten experiments were done by this method, as recorded in the accompanying table. Six were done in the cervical esophagus, and four were done in the thoracic esophagus. In none of these did leakage of the esophagus occur, but in three (two cervical and one thoracic) we failed to get sloughing of the esophagus after tying the ligatures because they were not tied tightly enough. A considerable amount of pressure must be exerted by the ligature. In our later experiments, we made it a practice to "saw" the ligature before tying it, thereby cutting through the muscular wall of the esophagus. There is little danger of cutting through the tough squamous epithelial lining of the esophagus by this procedure.

For the sake of brevity, these experimental results are given in the accompanying table.

Results in Ten Experiments

	Experiment Number	Days Between Operations 1 and 2	Adhesions Fascia-esophagus	Leakage	Slough	Stenosis	Killed, Days after Operation 2
Cervical	1	8	++++	0	+	+	12
	2	7	++++	0	0	++++	10
	3	4	++	0	+	++	16
	4	5	+++	0	+	+++	4
	5	6	+++	0	+	+	3
	6	7	+++	0	0	+++	1
Thoracic	1	8	+++	0	0	++++	7
	2	7	++	0	+	++	1
	3	7	+++	0	+	+	2
	4	7	+++	0	+	++	10

These experiments given in the table indicate that it is possible to remove a section of the esophagus without actually opening its lumen at the time of operation. The most important feature of the procedure is, therefore, that infection of the mediastinum by the contents of the esophagus is avoided, both at the time of operation and subsequently.

The two stage operation is simple. At the first operation, two ligatures and a sleeve of fascia lata are placed around the esophagus; at the second operation, the two ligatures are tied tightly and the two needle holes in the sleeve of fascia are closed.

STENOSIS

Our previous experiments indicate that stenosis may or may not occur following reconstruction of the esophagus with fascia lata transplants. We believe that it will be the rule for a cicatricial stenosis of some degree to follow this procedure. We have not dilated any of our experimental reconstructions with fascia lata since we wished to ascertain whether or not a stenosis would follow. Even if a stenosis should occur, and if we should be successful in removing the carcinoma of the

esophagus without a fatal mediastinitis, the patient would certainly be benefited by the exchange of a carcinoma for a stenosis.

Operation in one case of carcinoma of the esophagus was attempted by this method. The operation was not carried to completion on account of the extensive infiltration of the carcinoma. The diagnosis of carcinoma of the esophagus had been made nine months previously, and he had received three series of radium treatments to the carcinoma. With 0.5 per cent. procain, the intercostal nerves were blocked (after exposure) along the eighth, ninth and tenth ribs, left, and these ribs were resected for 10 cm. of their length, beginning about 2 cm. from their attachments to the spine. The carcinomatous esophagus was about 6 cm. from the cardia. After exposure of the esophagus, it lay deep in the wound and would have been more easily exposed if we had approached it from the right side of the spine instead of from the left side. The pleura was not entered; the procain anesthesia was very satisfactory until the esophagus itself was reached, when ten minutes of nitrous oxid-oxygen anesthesia was given. The carcinomatous esophagus was dissected from its bed, for about 8 cm., and fascia lata laid over two-thirds of its circumference. No ligatures were placed around the esophagus. There was no leakage or sloughing of the esophagus following this procedure. The case was considered as "inoperable," and nothing further was done. There was little shock following the operation. On the second postoperative day, the patient was able to sit up in bed and to enjoy a cigar. He died thirty-eight days later of a pneumonia and cachexia. Necropsy was refused.

CONCLUSIONS

1. By a two stage operation, it has been shown, experimentally, to be possible to remove a section of the esophagus by ligation without actually opening the lumen of the esophagus at either operation.

2. Infection of the mediastinum from leakage of the contents of the esophagus is eliminated by the procedure.

DISCUSSION ON THE PAPERS OF DRS. TOREK, EGGERS AND ALLEN

DR. WILLY MEYER, New York: I am sure that the members of our association and other colleagues interested in its work are happy that we now have three cases of resection of the thoracic portion of the esophagus with operative recovery, done by three fellows of our association. They are the only three in the world. Of course, we realize that only the beginning has been made; more will follow.

Dr. Torek has had the great pleasure of being able to report that his patient is alive eleven years after the operation. Dr. Lilienthal perhaps would have had the same satisfaction could he have been technically able to expose further the field of disease by the mediastinal approach, the roentgenograms having been somewhat misleading as to the exact level of the existing neo-

plasm. The primary formation of the skin flap to be inverted dictated the level in his operation. I had the pleasure of being present at Dr. Egger's operation, and saw the splendid technic. The work was thoroughly radical. I do hope he will be able after eleven years to show us his patient still alive.

Let us consider for a moment why we have not had a larger number of favorable immediate results after esophageal resection, many of us having been at this work since 1908. The reasons are many; I want to mention just a few. The principal cause in the beginning was that we did not know how best to operate, also, that we thought the experience gained in animal experimentation could be simply transferred to the human body. Today, we know how we must go ahead. I believe that the transthoracic approach with antethoracic transposition of the proximal esophageal stump gives the best chance for getting a favorable result. A further principal cause why we did not see more patients recover was that the patients came, and still come, too late. Let us all bear in mind—and we cannot talk enough and write enough about this point in order that every general practitioner, who usually gets these cases into his hands first, will grasp the importance—that almost 90 per cent. of all spontaneously occurring strictures of the esophagus in patients about and above 30 years are cancerous. The general practitioner must learn that, at least in the poorer classes, it is best to consider these patients hospital cases at once, for the sake of making the correct diagnosis, except when the patient refuses to enter. Let us all remember, that without efficient, prompt treatment we have 100 per cent. mortality, also that the frequently found squamous cell epithelioma of the esophagus is, so to speak, the most benign of the malignant neoplasms known in man.

The diagnosis of a beginning esophageal cancer is difficult. The roentgenologists made an effort, particularly in the early time of this work, some six to eight years ago, of going ahead and helping us by fixating the early diagnosis on the film. But it seems that their enthusiasm has quieted down. What they first tried to carry out has not been continued; this again for the principal reason that these patients come into the hospital by far too late.

Let us not forget, in this connection, that exploratory thoracotomy is at our disposal. All that is required at the time of exploration is to be ready for the major operation, should a malignant infiltration be found. The exploratory thoracotomy, as such, is a pretty safe operation.

Now, of course, the surgeons could not immediately present a series of recoveries after resection. So it occurred that the roentgen rays and radium quickly came in as a competitive treatment. Naturally, the general practitioners, not seeing their patients recover from the resection, said, "Why should we turn these patients over to the surgeon if the majority die from the operation?" And so they sent them to the roentgen ray specialist, particularly to the one working with radium. It certainly was hard luck for the modern operation for cancer of the esophagus that it came up just at the time that roentgen rays and radium rays were introduced in the therapy of deep seated cancer.

Before coming here, I received the program of the ninth annual meeting of the American Radium Society, which is going to take place in Chicago, June 9 and 10. Among other papers, the following is announced: "Third Report of Cancer of the Esophagus Treated with Radium" by Charles W. Hanford of Chicago. The abstract reads:

"This report covers observations of one hundred cases of carcinoma of the esophagus treated with radium. All cases of esophageal cancer are advanced

when they are first observed. Therefore, cures are very rare. Improvement follows treatment in a small percentage of cases. This improvement is not of long duration, as a rule.

If we can eradicate every part of the lesion, our results may be better. But we are working in a viscus where we are denied ocular demonstration of the position of our applicator; some portion of the lesion is liable to escape the lethal action of the rays, and the end-result is not as gratifying as when treating lesions in more exposed situations."

This certainly is an honest statement. What we need, at this moment, is that the radium specialists come forward, publish and present their cases of "cure" of esophageal cancer by means of radium, exhibiting at the same time microscopic slides by pieces cut out by the esophagoscopist to verify the diagnosis. I personally believe that the final result of their treatment is not what they had expected. In other words, the pendulum that had swung toward radium for many years will now, particularly after the report of another recovery from transpleural resection this morning, swing back to surgery. And why should it not?

In transpleural operations, we need differential pressure; in the mediastinal approach, it should also be on hand. We know from Tiegel's tests that 1 mm. plus-pressure of pure oxygen maintains life, when the pleural cavity is open. At present, the majority of surgeons are using gas and oxygen with the mask. It is the easiest way to operate. Every little while in the course of the operation the lung is totally inflated, because such thorough distention is the best stimulant for the heart by physical means known to us; otherwise, it is allowed to collapse. Tremendous progress has been made in carrying out this operation by carefully prepared team work.

One more word, in closing, regarding cancer of the cardia. In the majority of cases, the cancer of the cardia is not only a cancer of the cardia proper, but it involves also the lower portion of the esophagus. Last year, an interesting contribution to this chapter was given by Grégoire of Paris. He gains access to the very seat of the trouble, the cardio-esophageal segment, with the help of a plastic operation from a posterior (mediastinal) incision without interfering with the pleura and peritoneum. It certainly appears advisable to test thoroughly this type of posterior approach in cases of cancer of the cardia. I fear that the surgeon cannot help interfering with pleura and peritoneum.

Dr. Hedblom had the great satisfaction of seeing one patient with resection of the cardia for cancer pull through, the first of this kind in America. There are now six cases of resection of the cardia that have recovered from the operation, according to the literature of the world. I am sure more will follow.

In conclusion, let me say, that the American Association for Thoracic Surgery ought to have joint meetings with the American Roentgen Ray and Radium Society and compare notes with regard to final results, alleviations as well as cure. In that way, this important question of the best procedure of treating cancer of the esophagus would make further and more rapid progress.

Dr. Edward Archibald, Montreal: My experience in the last two years, since I took part in the discussion on Dr. Lilienthal's case, has not been such as to justify more than one or two remarks. In reporting cases and in estimating the value or the workability of a given method, it is surely time to make a clear distinction between the low carcinoma, down near the diaphragm, and the high carcinoma.

Dr. Torek is to be congratulated on the fact that his technic, in the hands of Dr. Eggers, can now register a second success. That is a unique achievement in this branch of surgery. Dr. Allen's résumé of the subject hit the nail on the head in emphasizing infection as the chief disadvantage of the majority of our methods. That, of course, is the great stumbling block. I don't believe that shock, by itself, is a serious cause of the still too usual death. I do believe, however, that acute pneumothorax, on top of a certain amount of shock, adds a great deal to the danger of the operation, and is frequently underestimated in that respect.

Dr. Allen's operation must have impressed us all very favorably by the ingenuity of his method in the avoidance of infection. He will probably be bothered with a stenosing scar, but he will likely avoid a death from infection.

Dr. T. C. Davison, Atlanta, Ga.: Statistics show that carcinoma of the esophagus occurs most often in the middle third, and less often in the lower third, and rarely in the upper third of the esophagus; on account of the inaccessibility which makes it anatomically almost impossible to remove without destroying or injuring certain structures in the middle third, on account of the relation to the aorta, to the vagus, and the pleura, this operation is quite difficult, and I think that Drs. Torek, Eggers, Lilienthal and Hedblom deserve a great deal of credit for blazing a way and progressing as they have in this most difficult operation. I think Dr. Eggers has improved on Dr. Torek's original technic, and there will be other improvements, of course, as we progress with this extensive and difficult operation.

These patients practically always come to the surgeon late. The two symptoms which drive them to the surgeon are inability to swallow solid food, and pain. Those are late symptoms, and, therefore, we do not have a fair chance at these patients. When they do arrive, we find, of course, that the carcinomatous condition is extensive, the inflammatory condition has attached, by adhesions, the esophagus to the surrounding structures which adds to the difficulty of this operation.

The Torek technic has its advantages, and, I feel, also its disadvantages. The great disadvantage, I think, is in that is is a very extensive operation to be attempted at one step. Carcinomatous patients are notoriously bad risks in the late stages, and many of them will die from shock. Probably there are some unsuccessful cases that have not been reported. We have our successful cases reported here, but there are, no doubt, many unsuccessful ones which are never mentioned.

I want to mention Dr. Hedblom's case in which he did an operation by several steps, first resecting the ribs, allowing the lower pleural cavity to collapse so that, when he did open his pleural cavity, there was not such an extensive collapse of the lung and not so much shock with his operation.

We look forward, of course, with a great deal of anticipation to the improvements on all of these operations in the near future.

Dr. Evarts A. Graham, St. Louis: I have watched the method described by Dr. Allen from its incipiency, and, from watching his experiments on animals, I feel exceedingly optimistic that it will be a method which will be directly applicable to the human being, and which will eliminate infection certainly in most cases in which it is tried.

Unfortunately, Dr. Allen did not go into a full presentation of the method. There is one part of it which is quite important which was omitted, and that is that his method of approach is an extrapleural approach from the rear. The pleura is not open, another safeguard against possibility of infection.

The question comes up whether or not there might be some danger of this necrotic mass lodging at the cardiac end of the stomach and not passing on into the stomach. I believe that it is quite possible that that might happen once in a while, but I do not regard that as a serious obstacle to the operation in itself when we realize the present terrific mortality which is present with the now used methods for operation for carcinoma of the esophagus. Suppose it does happen occasionally that the necrotic tumor lodges against the cardia and fails to pass, supposing it even happens once in a while that such a calamity as that would result in death, it would certainly be a minor event, it seems to me, in comparison with the almost inevitable causes for death which exist in the present methods for operating on the esophagus.

Dr. George J. Heuer, Cincinnati: In response to Dr. Archibald's request, I will briefly outline what Drs. Andrus, Bell and myself have been doing, the past winter, on this question.

It would seem that we have to devise different procedures for carcinoma at different levels of the esophagus. Our interest has been centered on those lesions at the cardiac end of the esophagus or at the cardiac end of the stomach; and, believing as Dr. Allen does, that infection has played the greatest rôle in the mortality following resection, we thought we might perhaps make conditions more favorable if we made the lower end of the esophagus an abdomnial viscus before attempting resection. This may be done by elevating the left half of the diaphragm, a procedure which has now been carried out on twenty-five or thirty dogs. It has been found possible to transpose the lower 8 or 10 cm. of the esophagus into the abdomen, and this procedure constitutes the first stage in the contemplated resection.

At the second stage done some time afterward, the abdomen is opened by a thoracic incision, the esophagus is resected and an anastomosis is made between the fundus of the stomach and the upper end of the divided esophagus.

This procedure has been carried out on dogs in a good many instances, and we can say that, for the dog, the operation is perfectly satisfactory. There has been, in two instances, I think, a stricture develop at the site of the anastomosis between the upper end of the divided esophagus and the fundus, but that is a complication which I think with experience can be avoided.

The procedure has the advantage that the dangerous part of the operation is done within the abdomen where the chances of infection are less. We have the omentum and other structures to protect our anastomosis. We have a cavity which I do not think is so likely to spread infection as is the thoracic cavity, and we have the additional advantage that it is perfectly easy to resect at the time—I mean granting that the patient's condition warrants it—the lesser curvature of the stomach.

We tried this procedure in one human case and moved the diaphragm upward without difficulty; the only trouble in the course of the procedure was that the esophagus was densely adherent to the aorta and to the pericardium. We made the mistake in this instance of freeing about 10 cm. of the esophagus, that is to say, we not only moved the diaphragm upward well above the level of the upper margin of the growth, but we also freed the esophagus from its bed. The patient developed signs of infection forty-eight hours after operation and died, and at necropsy the part of the esophagus which had been mobilized had undergone gangrene at one point with perforation and leakage into the pleural cavity. So far as the transposition of the diaphragm was concerned, the result was perfectly satisfacory.

Since that event, we have not mobilized the esophagus as a preliminary procedure, and should we get a second favorable case, we would transplant the diaphragm as a primary procedure and subsequently mobilize the esophagus, resect the tumor and perform our anastomosis all at the second stage.

Dr. Nathan W. Green, New York: The diagnosis of carcinoma of the esophagus is comparatively simple and easy, if one is at all familiar with the condition. The fact that a person out of a clear sky has dysphagia, and this is repeated and shows a tendency to be permanent, in about nine cases out of ten, should point toward a carcinoma of the esophagus.

At one of the hospitals with which I am connected there is a great tendency to use radium. To try it out is the purpose of the hospital. In the treatment of carcinoma of the esophagus, the men using radium feel that they have been able to accomplish very little; this also is true of the roentgen ray. Consequently, it is for us to redouble our efforts toward surgical intervention.

Now, with Dr. Torek's successful case, Drs. Eggers and Hedblom's one case and Drs. Heuer and Allen's very ingenious method, it would appear that we have a phalanx of five strong men who will help us advance the line in this defense quite materially.

Of course, the keynote of the recovery from the operation for carcinoma of the esophagus is the prevention of infection, and Dr. Torek has hit the nail on the head when he has done the exenteration of the esophagus.

I am interested to see in Dr. Eggers' case that the cut vagus nerves in this human demonstration have not resulted in spasmodic closure of the pylorus. Of course in the lower animals where the physiology is more vigorous, the section of the vagus nerves paralyzes the action of the pylorus; one can see that in cats and dogs. But, in this case and in a case of midgastric resection that Dr. Downes did some time ago, not for carcinoma but for an ulcer, the pylorus was not paralyzed, so it did not remain constricted. Of course, the vagus nerves act and cause relaxation of the pyloric muscle which otherwise remains closed and sphincteric.

In the results of Drs. Torek and Eggers, it seems to me that the patients have remained so comfortable that it will be hard to improve on their condition; the tube that connects the end of the esophagus with the stomach can be discontinued and cleaned out if there is any stoppage.

The gastrostomy that Dr. Eggers did appears to be very helpful in cases of emaciated patients with starvation. The gastrostomy may be one of two types: either of the simple Kader-Senn type, which will close up as soon as the tube is removed, or shortly after, or a permanent type of the Janeway variety, which will remain closed when not in use but never closes permanently without some effort to closure.

Another point I should like to bring out is that the squamous cell epithelioma of the esophagus is very close in its metastases. I have seen many cases of epithelioma of the esophagus and comparatively few that have come to necropsy in the squamous cell type have shown metastasis at any distance. On the other hand, the closures of the esophagus from new growth of the cardia and near the cardia may be due to the cylindric cell type, and I am inclined to think that those form more rapid metastases.

Dr. A. L. Lockwood, Toronto: I want to say one word along the line that Dr. Allen has been working on. In 1919, I had the idea that it was possible to use an epithelized graft of skin, and after I had done it on a good many cadavers I tried it on ten dogs but had no success with it.

I must confess that I feel very humble today about this question of high up, annular carcinoma of the esophagus, especially after Drs. Eggers and Torek's success. I saw a patient, in the early days of 1919, with a high up, annular type of carcinoma of the esophagus, in London. I had first planned to do Dr. Torek's operation on the patient, but I had been so much impressed by the possibility of swinging in an epithelized graft of skin that I thought I would try it — excise the growth and form a new tube in the epithelized graft of skin. The growth was at the junction of the upper and middle third of the esophagus. I did not resort to quite the same incision that Dr. Torek did. I went through the third interspace and then resected the rib above and below, through the costal margin. The growth measured about 2.5 cm. I had previously prepared a wide flap of epithelized skin, depilated first and then epithelized it. I was persuaded, however, to follow the technic of Trotter, the senior surgeon of Kings College Hospital; he has attacked several of these patients — none of them lived, but he has used all fascia grafts. He made a tube of this graft and put it in without leaving the growth, as Dr. Allen suggested. I put in a fascial graft. The patient began to have an elevation of temperature the next day and died, the seventh day, of mediastinitis. I do feel that if we had put in the epithelized skin graft we might possibly have had a success.

Dr. William H. Stewart, New York: Some years ago, with the aid of Dr. Crump, I brought out the sausage skin method of examining the esophagus. It is especially valuable in early cases. Unfortunately, the method has fallen into disuse on account of the advanced lesion present when first we see these cases. Most of them can be recognized by the ordinary method of roentgen-ray examination. If we can educate the medical men to the possibility of surgical relief in cancer of the esophagus, I feel sure that we will succeed in getting cases earlier. If we do, then again comes the sausage skin method. It is a very simple procedure; one simply desensitizes the patient's pharynx with a bougie for two or three days until one is able to swallow a piece of ordinary sausage skin closed at one end, which passes down beyond the obstruction; this is filled with bismuth subcarbonate held in suspension in mucilage of acacia and water. This is followed by the roentgenographic examination.

The beauty of this procedure is that it not only accurately locates the tumor but also, in addition, gives exact information to the surgeon of the condition of the esophagus, through the growth and beneath as well, which is very important.

Dr. Porter P. Vinson, Rochester, Minn.: I do not believe that we should consider all or even 50 per cent. of these cases of carcinoma of the esophagus operable. The early diagnosis of cancer of the esophagus will, of course, help us in getting favorable cases, but certainly many of these patients have advanced lesions before symptoms develop. Even though dysphagia is the earliest symptom, carcinoma of the esophagus is probably, in the majority of cases, advanced and inoperable before dysphagia begins. As long as a patient has a lumen in the esophagus that will permit the passage of a 30 French sound, he can eat any type of food without trouble, so that the lumen of the esophagus must be considerably narrowed before the patient has symptoms of dysphagia. The lesion is out of sight, and the patient unconscious of it.

We often see patients with metastasis to the cervical glands who have no symptoms referable to the esophagus. Such conditions are hopeless so far

as surgery is concerned. In order to make early diagnoses, the public must be educated, as it has been educated with regard to carcinoma of the breast. In this event, positive diagnoses could probably be made six or seven months earlier.

Our experience differs from that of Dr. Meyer, who believes that 90 per cent. of patients with dysphagia have carcinoma of the esophagus. Less than 50 per cent. of the patients who come to the Mayo Clinic complaining of dysphagia have cancer. At least 25 per cent. have cardiospasm, which is often diagnosed cancer. A great many patients with benign stricture come to us with the diagnosis of cancer, and radium and roentgen-ray treatments have been given.

The early cases in which there has been no pain and in which the symptoms are of short duration should be chosen for operation. Pain is an ominous symptom and is very rare in the early stages of cancer of the esophagus; it occurs early in cardiospasm. There are early cases of cancer of the esophagus in which the diagnosis can be made with comparative certainty by means of the esophagoscope, but cannot be verified microscopically because normal mucous membrane has piled up over the lesion in such a way that only inflammatory tissue is obtained in a specimen. Hence, certain patients will have to be operated on without a positive microscopic diagnosis.

In treating cases of malignant strictures in the clinic, it has been found that mechanical dilatation offers more palliation than any other form of treatment. In our experience, radium is of no value; the roentgen ray may be beneficial in occasional cases; we have one patient who has improved following deep roentgen-ray treatment. I would suggest mechanical dilatation and roentgen-ray treatment in handling inoperable cases. Life has been prolonged for three years following a single dilatation of a malignant stricture, and it is a relatively safe procedure.

Dr. Carl A. Hedblom, Rochester, Minn.: I should like to emphasize the value of the posterior extrapleural approach to the thoracic esophagus. I think any one who has not made this exposure or who has not seen it can scarcely appreciate what a good exposure it affords, particularly to the middle part of the esophagus behind the root of the lung, the part that is, of course, the most difficult to expose by any other method.

I have explored in many cases in which I did not attempt resection, because, as Dr. Vinson has said, so many of the cases are relatively late.

Dr. Lilienthal reported his experience, two years ago, and I would fully agree with all that he said with the exception that, in my experience, the right exposure is much better than the left. One avoids the obstruction of the aorta on the right side and the arch of the azygos affords an excellent landmark by which to identify the esophagus. It can be done in a few moments.

One point that I think is of a good deal of importance is the fact that an infection of the mediastinum following resection of the esophagus is by no means necessarily fatal. I have had three cases of infection of the mediastinum with an accumulation of from 400 to 1,000 c.c. of pus, and all three patients got well following irrigation of the cavity.

I think Dr. Heuer's suggestion a particularly promising one in dealing with the cases of carcinoma involving the cardiac portion of the stomach, which I think belong in the domain of thoracic surgery. If one can get a segment of the thoracic esophagus transposed below the diaphragm, it will greatly facilitate later anastomosis.

DR. FRANZ TOREK, New York: As regards diagnosis, Dr. Vinson has been telling us to pick out the early cases. We would be most happy to do so. However, if we had refused to operate in the cases with dysphagia, of which he says that they are too late for operation, we wouldn't have operated in any.

As for the early symptoms, Dr. Eggers mentioned that his patient had a girdle pain as the first symptom. That was very interesting to me because the patients I found who had an early symptom preceding dysphagia had a sensation of tightness in the neck. If this is present in a person old enough to be of the cancer age, one should remember that it might be an early symptom of carcinoma. As regards the sausage skin method, it may be used in the early cases but in the late cases it is impossible for the patient to swallow the skin.

Not very much has been said about esophagoscopy. Two years ago, I spoke very strongly in favor of esophagoscopy, but since then I have changed my mind, as I had two patients die from injuries caused by the esophagoscope, although the work was done by an expert.

One speaker touched the subject of local versus general anesthesia. I shouldn't want to be dogmatic on either the one or the other. Either may be used. I prefer the local anesthesia, and the fact that a great deal of time is used in administering it before the operation begins does no harm to the patient.

Another point that has been brought up is that of drainage. Here again I don't wish to be dogmatic either pro or con. One may have good success with or without drainage, and one may have failures with or without drainage; I have had a number of failures with drainage; consequently, drainage has not been a safeguard in my hands. Dr. Eggers told us that his patient drained 200 c.c. the first day and 50 c.c. the second and third days; quite likely it would not have made any difference if those 300 c.c. had remained in the cavity undrained. One may be successful either way. Drainage is not the important question.

I have been very much interested in the methods of technic that have been shown here. My own method for carcinoma of the cardiac end of the stomach, which I published in April, 1915, was to do exenteration of the esophagus above the carcinoma and later a partial gastrectomy from below.

As regards the side from which the esophagus should be approached in the fascia flap operation, it is absolutely imperative to go in on the right side because one cannot do it from the left side, unless the entire operation can be performed well below the arch of the aorta. Higher up, the arch of the aorta is in the way, if the approach is attempted from the left side.

There are two other points that Dr. Allen brought up that require some elucidation. One is that of shock. He believes that shock is going to be diminished by this operation. The greatest amount of shock in the operation is apt to be caused during the separation of the esophagus from behind the arch of the aorta. When that part is done, the rest of it, in my estimation, is comparatively easy and not very likely to increase the shock.

The other point was that of infection. As regards that, I have no great fear of infection by my method.

DR. CARL EGGERS, New York: I should like to call attention to the fact that, up to the time of Dr. Torek's first successful case, patients who were operated on for carcinoma of the esophagus died of certain well known causes, of which leakage was one of the most important. His method was devised

to prevent death by these causes. If the principles he has advocated are correct, it should be possible to save more patients by this method. Personally, I have been much impressed with the soundness of the principles underlying the operation, and have therefore followed them, and I believe that the value of my case lies right here, in putting Dr. Torek's operation on a more solid footing. His patient is no longer just a chance patient who got well; another patient has now successfully been operated on by the same technic.

Knowing that a certain method will give positive results in suitable cases should encourage us to work along that line with enthusiasm. We must not be satisfied with having two cures; we should have more, and we should have them soon.

DR. DUFF S. ALLEN, St. Louis: In regard to infection, I might say that we arrived at our conclusions by reading literature, it is true, but we arrived at it in an experimental way also.

We tried to sew fascia lata in defects in the esophagus, in the thoracic portion, at a one stage operation. Regardless of the size of the opening, the experimental animals died within from twelve to twenty-four hours, always with marked leakage, so I am not at all surprised to know that even a small defect would leak.

In regard to the skin flap, I might say that we are working on a method now, several methods, of trying to sterilize a skin flap. So far, these methods are so embryonic that I won't mention them any further.

As for cases being operable, as Dr. Vinson brought out, Dr. R. Walter Mills, whom you all know, was under the impression that carcinoma of the esophagus was not only the most benign carcinoma, and metastasized later than any other gastro-intestinal carcinoma, but also that it was one of the earliest diagnosed; that was his experience.

Certainly, we employ the esophagoscope on every one of these cases. A case with dysphagia is immediately esophagoscoped, and small ulcers are picked up that way. Up to the present time, the treatment has been mainly radium and roentgen ray.

The point that Dr. Graham brought up about the possibility of this necrotic area or necrotic portion of the esophagus lodging at the cardia of the stomach may be well worth consideration. However, I have never felt that that was a great hazard. You see these patients have had a gastrostomy before, and it is very easy to get up into the lower portion of the esophagus with the esophagoscope, and I should think we could take this thing out if it happened to lodge there.

Dr. Heuer's idea of making the esophagotomy in the abdominal cavity I think is well worth consideration. There is one thing that we want to remember about the esophagus, though, and that is the covering of the esophagus. The intestines further down have a peritoneal covering; not so the esophagus. I think, then, that the proper tissue to expect to grow to the outer wall of the esophagus would be fascia, and I think that if we have trouble in leakage in the abdominal cavity, the same as we do in the thoracic cavity, undoubtedly it would not be so serious.

In regard to the length of the section of esophagus resected, it is true that we haven't resected a very great portion and a very long portion of the esophagus, and we know that we would have to use more fascia and resect a longer portion if we were to resect the carcinoma of the esophagus in the human being. The fact that we haven't resected more in the dog is because the fascia lata of the dog is small, and we have resected just as much

as the fascia lata would cover over. Those of you who have seen the fascia lata of the dog know that it is about 2 inches (5 cm.) wide, so we resected as much as possible and got away with it.

Infection of the suture is a possibility, but it hasn't occurred. When they are tied at the second operation, the knot is pushed inside and then this little needle hole is closed up, and the new connective tissue is brought over that. I think that what happens there is that, before we have gotten our slough, that has sealed itself up. We are contemplating a method now of getting this tied from the inside of the esophagus, so that we don't even have to have the little needle holes.

CAUTERY PNEUMECTOMY FOR CHRONIC SUPPURATION OF THE LUNG

A REPORT OF TWENTY CASES *

EVARTS A. GRAHAM, M.D.

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Of all the methods that have been used in the treatment of chronic lung suppuration, the one which, theoretically, would seem to be the most nearly ideal is the complete extirpation of the diseased tissue. Unfortunately, however, the ordinary resection of a lobe of the lung in a condition of suppuration has proved itself to be an extremely hazardous procedure. In a recent article, I [1] showed that the mortality in the forty-eight reported cases of lobectomy for bronchiectasis was 52 per cent. There is no doubt that many cases will yield to measures which accomplish compression or shrinkage of the lung tissue, but many others will not. Moreover, even in those patients who are symptomatically improved but who still retain their infected tissue, there is always danger of the occurrence, at some time, of a serious cerebral complication, a severe pulmonary hemorrhage or an extension of the inflammatory zone. Actual removal, therefore, of the diseased tissue is the desired aim if it can be accomplished safely. In this respect, the situation is analogous to chronic inflammation elsewhere, as in the gallbladder or appendix.

Necropsy examinations have revealed the fact that almost all the deaths from lobectomy in cases of suppuration have been due to infection. These findings are in accord with what might be expected when the necessary method of performing the operation is considered. For, in order to remove a lobe, which is usually adherent, it is necessary first to mobilize it by freeing the adhesions. This necessary mobilization at once causes the production of large areas of raw surface without the protection of the pleural covering. These areas, furthermore, usually lead to regions that are particularly vulnerable to infection, namely, the pericardium and the mediastinum. When the bronchial stump opens up, therefore, highly infectious material is poured out in contact with these vulnerable areas.

It occurred to me that perhaps the diseased tissue could be removed with a minimal danger of infection by means of the actual cautery.

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* From the department of surgery, Washington University Medical School and Barnes Hospital, St. Louis.

1. Graham, E. A.: The Surgical Treatment of Bronchiectasis, Arch. Surg. 6:321 (Jan.) 1923.

A description of the operation, as used in three cases, was reported last year.[2] It seemed desirable to report on a more extensive experience with it since that time. Up to the present time, I have carried out extensive cauterizations in twenty cases of chronic lung suppuration. In these cases, the condition has been present in the various individuals for periods of from six months to twenty years. They were all totally incapacitated, and many of them had been bedridden for several years. Some of them represented, therefore, the most extreme effects of chronic pulmonary suppuration. The results are summarized in the accompanying table.

Results From Cauterization in Twenty Cases of Chronic Lung Suppuration

		Per Cent.
*Free from symptoms and completely healed	4	20
Free from symptoms but with remaining bronchial fistulas	6	30
Marked improvement but with some cough still present	3	15
Improved, still in progress	2	10
Not heard from	1	5
Dead	4	20

*At the time of the correction of the proof of this article in October, 1924, two more patients (Cases 9 and 14) are free from symptoms and completely healed, making a total number, therefore, of six, or 30 per cent.

Of those who are classed in the table as "free from symptoms but with remaining bronchial fistulas," all are well except for the slight annoyance of the fistula. This merely necessitates the wearing of a small dressing over the fistulous opening which is changed by the patient as often as desired. The secretion from it is about the same in character and in amount as that normally from the nose, and it causes practically no inconvenience to the patient. It is possible also that some of these fistulas will close spontaneously later. In reality, then, ten patients, or 50 per cent., can be considered as well. Of the three who are listed as showing "marked improvement but with some cough still present," two have definite evidence of involvement in another lobe of the lung than the one operated on, a fact which probably accounts for the persistence of some of the symptoms. In these cases, the lobe which showed the most marked pathologic change was the one selected for the cauterization. It is possible that, at a later date, an operation will be carried out also on the other diseased lobe. The two patients

2. Graham, E. A.: Pneumectomy with the Cautery: A Safer Substitute for the Ordinary Lobectomy in Cases of Chronic Suppuration of the Lung, J. A. M. A. **81**:1010 (Sept. 22) 1923.

still in progress have not yet been discharged from the hospital, but their condition is distinctly improved. The deaths have all been from what have seemed to be accidental causes, three from cerebral complications, and one from pneumonia in the other lung after the operation. Of the cerebral complications, one was suppurative meningitis occurring three weeks after the last cauterization, and the other two were sudden deaths with general convulsions occurring soon after operation, apparently from cerebral embolism. There has been no death from infection of the mediastinum or pericardium. This incidence of three cases, or 15 per cent., of cerebral complications is apparently about the same as that which occurs spontaneously in this type of condition. For example, Tuffier [3] has reported brain abscess occurring as a complication in six out of forty-five cases of lung suppuration, an incidence of 13 per cent. This total mortality, therefore, of four cases, or 20 per cent., can probably not be ascribed entirely to the operation. But, even if it were due to the operation, it is less than one-half the mortality following the ordinary lobectomy, and the percentage of patients who have been completely relieved of their symptoms (50 per cent.) is very much higher than in the published results of lobectomy. In fact, a total of 75 per cent. of all my patients are known to be either entirely well or improved. I know of no other therapeutic procedure for these desperate chronic suppurations of the lung which can give so favorable a record of results.

In a recent article, Hedblom [4] has recorded his experience with the operation of graded thoracoplasty in ten cases of unilateral bronchiectasis. His results are better than those previously obtained by this method. He had no deaths, but it is uncertain if he obtained any final complete relief. By this statement, I mean that the patients still possess their diseased tissue, and experience has shown that, in this type of case, even long remissions may be followed by a reappearance of symptoms; and there also is always present the danger of cerebral embolism, pulmonary hemorrhage and extension of the infection. To quote from his summary:

> Six patients have good general health. Three of these are practically free from symptoms; three raise from 30 to 60 c.c. of sputum in twenty-four hours, and one has symptoms and signs suggesting extension of infection to other portions of the lungs, but raises only about 60 c.c. of sputum in twenty-four hours. None has shown disability or marked deformity. Three patients are still convalescent.

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3. Quoted by Sauerbruch, F.: Chirurgie der Brustorgane, Berlin, Julius Springer, **1**:569, 1920.

4. Hedblom, C. A.: Graded Extrapleural Thoracoplasty in the Treatment of Diffuse Unilateral Bronchiectasis, Arch. Surg. **8**:394 (Jan.) 1924.

It would appear that none of the patients is entirely free from symptoms. He states also that he has performed a graded thoracoplasty for abscess of the lung with complicating bronchiectasis in eight cases, of which three died "after operation" and one after he returned home. In two cases, "remarkable improvement, approximating cure" occurred. If all the eighteen cases, therefore, of chronic lung suppuration in which a graded thoracoplasty had been done were grouped together, not only would the mortality be higher but also the results less impressive than in the series of cautery pneumectomies which I have summarized in the table. In my series I have not distinguished between the clinical diagnosis of chronic abscess and of bronchiectasis because, in any case of chronic suppuration, both conditions are present to some degree, although, in some cases, the abscess feature predominates and, in others, the bronchiectasis. Drennen [5] has recently reported a very successful result in a case of chronic lung abscess treated by cautery pneumectomy, although he seems to have done more at one time than is usually advisable. I have also had personal reports from other surgeons of three other cases treated successfully by the method used in my cases.

The operation of cautery pneumectomy can be made to combine the three cardinal principles of the surgical treatment of chronic pulmonary suppuration, namely, drainage, compression and extirpation. For this reason, it would seem to have a wider applicability than any other method so far proposed. Again, the fact that it can be performed in multiple stages with relatively little disturbance to the patient makes it suitable even in patients whose vitality is very low. It can also be carried out when other methods, such as a thoracoplasty, have failed.

At the first stage, the field is exposed by turning up a flap of skin and muscle and the removal of three or four ribs for distances of about 4 inches (10 cm.). The ribs are removed by subperiosteal resection, and the periosteum is then peeled off the pleura with the intercostal bundles including the intercostal muscles, vessels and nerves. This procedure, in itself, permits an appreciable amount of compression of the affected lung tissue, although of course not to the same extent as in the Sauerbruch thoracoplasty. The exposed ends of the ribs are packed with bone wax to minimize the danger of osteomyelitis, and the adjacent intercostal nerves are injected with alcohol to destroy sensation in the wound. The diseased portion of the lung is now in view, covered by pleura to which it is usually densely adherent. It is essential, before proceeding further that the lung should be firmly adherent to the pleura. Otherwise, after one has gone through the pleura, the lung will retract itself away, and an empyema will result.

5. Drennen E.: The Use of the Cautery in the Treatment of Abscess of the Lung, Ann. Surg. **79**:639, 1924.

To determine definitely whether or not the lung is adherent, the pleura should be opened. If the lung is found not to be firmly adherent, adhesions can be created by any of the standard methods, such as suture and gauze packing against the pleura. If, while making the investigation, the lung has become retracted away from the pleura by an inrush of air into the pleural cavity, it will be found best then to close the pleura and to postpone any further operative procedure until after the lung fills the pleural space again. At that time, adhesions can be produced as suggested above. After the exposure has been made, the first stage of the operation can then be terminated, and, in patients who are bad risks, it is well not to proceed further at this stage. No sutures are placed in the flap.

After the lapse of a few days, the first cauterization can be done; or, if the patient is in good condition and if adhesions are known to be present between the lung and the parietal pleura, the first cauterization can be performed at the time of making the exposure. With a large soldering iron heated to a red heat, an excavation is then made into the lung tissue. If an old drainage tract exists, it is well to begin the cauterization by plunging the hot iron into the sinus and to work out from that. Other abscesses are sometimes found in this way, lying close to but not communicating with the main drainage track. In the cases in which the pathologic condition is chiefly that of a chronic abscess, the whole abscess cavity together with other smaller abscesses may be cauterized out in this way. In those cases, however, in which the chief pathologic condition is bronchiectasis, the first object sought after is to provide multiple drainage openings by exposing a large cross section of the bronchial tree. After the slough has separated there are sometimes as many as twenty or thirty bronchial fistulas, from each of which pus exudes, To accomplish this, the effort is made to begin by burning over a fairly large surface instead of by burning deeply into the lung. As a rule, the better the drainage established in the cases of bronchiectasis, the more rapid will be the convalescence. Repeated cauterization should not be done too soon. My experience has shown that it is better to wait about three weeks before repeating the cauterization. Indeed, in some cases, I have found that much less cauterization was actually necessary than had been originally supposed, if several weeks of free drainage through multiple bronchial fistulas had been permitted. There is, undoubtedly, some tendency in even the long standing cases for the diseased tissue to heal if given an opportunity by providing adequate drainage of the pus. This is in accord with the experience of Willy Meyer [6] with his "lip fistula." The establishment

6. Meyer, Willy: Observations on Lung Suppuration and Its Treatment, Arch. Surg. **6**:361 (Jan.) 1923.

of many bronchial fistulas by cross section of a relatively large part of the affected lobe certainly provides better drainage than can be accomplished in any other way. Moreover, the effect of the heat from the cautery is transferred to a variable distance away from the place with which it is actually in contact, and this effect doubtless leads to fibrosis of relatively large areas of the affected lobe and the simultaneous destruction of much secreting surface. The questions, therefore, of how much and how often to cauterize will be determined by the progress of the case after the first cauterization. In some of my cases, one cauterization has been sufficient and, in others, as many as six or eight have been required. The best rule to follow is that it is better to do too little at a time than too much. If hemorrhage from a large vessel occurs, it may be controlled by packing, and the operation is terminated. Two or three days later, the packing can be removed safely. I have never found any difficulty in controlling hemorrhage by packing, even if a large branch of the pulmonary artery has been severed. This is doubtless because the blood pressure in the pulmonary artery is only about one sixth of that in the systemic circulation. The control of hemorrhage is much easier if the exposure is generous. It is unwise and dangerous to attempt an extensive cauterization through a small opening. Moreover, by working in a small opening, one of the main purposes is defeated, namely, the creation of very free drainage by making a large cross section of lung surface and thus permitting drainage from many open bronchioles. After the operation, there is no specimen to show, as only charred fragments are removed.

Certain immediate effects of the cauterization are usually observed. While the cautery is actually in contact with the lung, there is frequently a period of apnea which may last for about 30 seconds, even after the cautery has been removed. Smoke may be exhaled from the nose and the mouth, and, if hemorrhage has occurred, there may be a little bloody expectoration. There is no shock, and the patients look as if no operation had been performed on them. For the first twenty-four or forty-eight hours, there may be either a marked diminution or a complete absence of cough and sputum. I am not certain about the explanation of this mechanism. It may be due merely to an abolition of the cough reflex. Generally, on about the third day, the patient develops a little fever and complains of malaise, loss of appetite and even sometimes of nausea and vomiting. These symptoms are apparently due to intoxication from the burnt tissue, and they furnish another reason for not performing too extensive a cauterization at one stage. These symptoms of intoxication are likely to persist until the slough has separated, usually about the tenth or twelfth day. Coincidently with the appearance of these symptoms, the cough and sputum may be increased and

may actually become greater than before the operation. The most probable explanation seems to lie in the pneumonitis of lung tissue adjacent to the cauterized area and created by the irritation of the cautery. Later, as the slough separates and as the open bronchioles begin to discharge pus to the outside, the cough and sputum diminish, the fever disappears, the appetite returns and the patient feels well.

The danger of severe postoperative hemorrhage after the slough has separated is a question that has been raised by some surgeons. The danger doubtless exists, but I have never seen any postoperative hemorrhage from the lung itself in any of my cases. The explanation doubtless lies in the low blood pressure of the pulmonary artery. In one of my cases, postoperative hemorrhage occurring from an intercostal artery was stopped by Dr. Allen by placing a clamp on the bleeding point. It is conceivable, of course, that occasionally hemorrhage might occur from a bronchial artery, since these arteries are part of the systemic rather than the pulmonary circulation; but the bronchial arteries are usually very small and, for that reason, they should not constitute a serious menace. Schlaepfer,[7] however, in his recent experimental study of the effects of ligation of the pulmonary artery has found that the bronchial arteries frequently become enlarged, and it is conceivable that they might become enlarged in cases of bronchiectasis. If the ligation of a branch of the pulmonary artery to a lobe in the human being should prove to be as easy and simple an operation as Sauerbruch[8] claims it to be, then the danger of hemorrhage could practically be eliminated in the operation of cautery pneumectomy by first performing a ligation of the corresponding branch of the pulmonary artery. If ligation of the corresponding pulmonary vein should also be performed, then the danger of cerebral embolism would also be practically excluded.

Auxiliary measures in the treatment of the chronic lung suppurations are important. Heliotherapy, when systematically carried out, is of great help. We have found the aspiration of the open bronchi also of considerable value. By means of a vacuum pump, gentle suction of the open bronchi is carried out daily or twice daily by the nurse. The drainage is materially increased in this way. This procedure has many advantages over bronchoscopic drainage in that it is more effective and there is no inconvenience to the patient.

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7. Schlaepfer, K.: Fibrosis of Lung Following Ligation of Pulmonary Artery, Combined with Phrenicotomy and Following Partial Occlusion of Pulmonary Veins, Arch. Surg. **6**:358-360 (Jan.) 1923.

8. Sauerbruch, F.: Chirurgie der Brustorgane, Berlin, Julius Springer, **1**:587, 1920.

REPORT OF CASES

CASE 1.—M. A., a woman, aged 40, admitted to the hospital, Jan. 2, 1923, after a previous admission, Aug. 30, 1922, had had a tonsillectomy, with ether, six weeks previously. About ten days later, she began to cough and to raise foul sputum. A diagnosis of a lung abscess in the right hilum region was made. There was foul sputum, about 500 c.c. daily. There were no tubercle bacilli, no blood was present in the sputum.

Pneumothorax was tried by Dr. Singer with indifferent results. In all, three injections of air were made; 500 c.c., September 7; 975 c.c., September 9, and 1,020 c.c., September 14. September 15, an exploratory operation was made in the eighth interspace. A cyanotic indurated mass, about the size of a lemon, was found in the right lower lobe in the posterior mesial aspect.

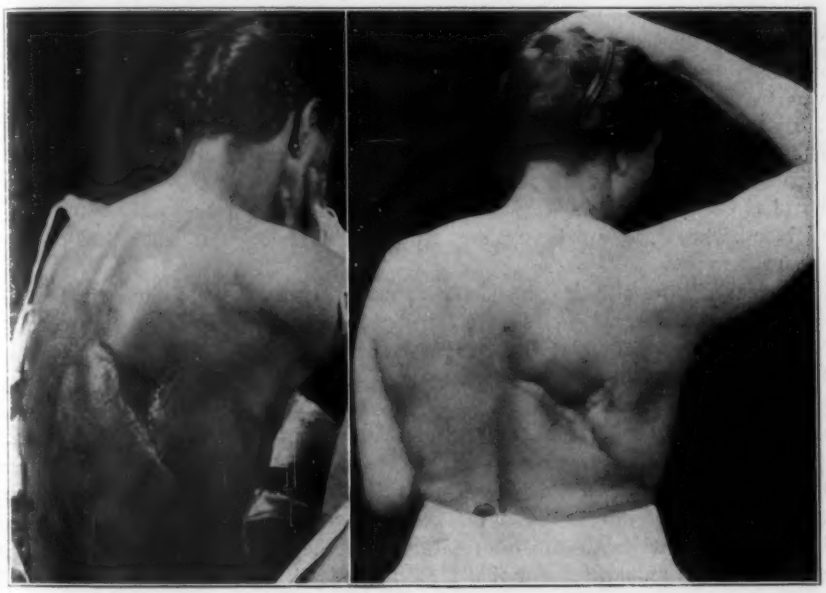

Fig. 1 (Case 1).—Twelve days after cautery pneumectomy; fifteen months after cautery pneumectomy; wound entirely healed, except for a small bronchial fistula, and the patient is entirely free from symptoms. Only a little mucus is discharged from the fistula; marked improvement in patient's nutrition.

The lung was sutured to the parietal pleura after resection of the eighth rib. September 18, the lung was examined with a needle in various directions through the adhesions. No pus was found. Before puncturing the lung, 200 c.c. of bloody fluid were aspirated from the pleural cavity. October 4, drainage of the empyema was done. Thick foul pus was removed. October 26, tube drainage of the lung abscess near the hilum was begun. Approach was made through the old area of adhesions after the removal of the seventh rib. There was free drainage of pus. A bronchial fistula was also observed. Improvement after this was not satisfactory. The patient still had fever of from 100 to 100.5 F. (38 C.). Cough and expectoration were still present, although less than before.

Jan. 18, 1924, an incision was made around the old sinus tract. The chest wall at this place contained fused ribs resembling an armor plate which were removed with a moderate amount of hemorrhage. The pleura was thickened to about one-eighth inch and was firmly adherent to the lung. It was dissected free from the lung for about 20 sq. cm. With the cautery, the mass of lung immediately beneath the exposed portion was removed. Another abscess was discovered containing 5 c.c. of thick foul pus. Many moderate sized bronchi were exposed. Convalescence was uneventful. The temperature was normal on the twelth day. There was a rapid gain in weight. The patient was discharged, March 30, and is perfectly well at present except for a fistula. This fistula causes no annoyance to the patient, and she prefers to keep it rather than undergo another operation for its closure.

Case 2.—G. C., a woman, aged 27, admitted, Jan. 28, 1923, had suffered the onset in September, 1921, with headache, chill and fever. She felt sharp pain in the right lower quadrant of the chest. She has had foul sputum, often with hemoptysis, since then. She lost 55 pounds (24.9 kg.) in a year. A diagnosis of lung abscess of the right lower lobe was made. Artificial pneumothorax was tried unsuccessfully. Sept. 14, 1922, at a former admission to the Barnes Hospital, the lung abscess was drained after removal of the ninth rib. Forty cubic centimeters of pus were obtained, and a tube was inserted. Convalescence seemed satisfactory at the time of discharge. Jan. 28, 1923, she reentered the hospital because about a week previously she had coughed up over a teacupful of blood, and there had been about 80 c.c. of sputum. Pus was still draining from the wound.

January 31, an elliptical incision was made around the old sinus tract. The seventh, eighth and ninth ribs were resected for a distance of 3 inches (7.6 cm.). There had been fusion of the ribs into one bony plate. A large cavity was burned out, including the old sinus tract. The sinus led up to a place very close to the hilum. There was very little hemorrhage. The patient was discharged, March 29, with only slight drainage from the wound. A small sinus was present through which air escaped on expiration. There was marked improvement in the general condition, and almost no cough or sputum. It has been impossible to follow this patient's case. Letters have been returned by the postmaster with the information that the patient has moved and the new address is unknown. For that reason, she is classified as "not heard from."

Case 3.—G. L., a white woman, aged 26, married, admitted, Jan. 3, 1923, on Aug. 28, 1922, had a tonsillectomy elsewhere under ether anesthesia. Nine days later, she began to cough, and had fever and pains in the right side of the chest and the right arm. The pain was sometimes relieved by coughing. The sputum often contained blood. She had lost 17½ pounds (7.9 kg.) in the last five months. She had been taking 1.5 grains (0.1 gm.) of morphin daily for several weeks.

Physical and roentgen-ray examinations showed evidence of abscess of the right lung near the hilum, on the level of the sixth, seventh and eighth ribs, posteriorly. The left lung apparently was normal. There were 300 to 500 c.c. of sputum daily, of very foul odor. No tubercle bacilli or spirochetes were found in repeated examinations. Elastic tissue was abundant. The complement fixation test for tuberculosis was negative. The Wassermann reaction was negative. Albumin was sometimes present in the urine. Hemoglobin was 80; red cells were 4,900,000; leukocytes, 15,500. The temperature was 38 C. (100.4 F.).

A pneumothorax was done by Dr. Singer, January 9. There was no reaction and no appreciable effect. Again, January 11, he introduced 400 c.c. of air. Following this, the sputum was reduced to 150 c.c., and the cough was less. January 15, the vital capacity was 1,700 c.c. On the same day, another pneumothorax was done with the introduction of 700 c.c. of air. January 21, she was permitted to go to her home in the city. She felt much better, and her sputum now amounted to only 100 c.c. January 29, she was readmitted to the hospital because she felt worse. February 2, 200 c.c. of foul thin pus were aspirated from the right pleural cavity. This empyema was considered to be due to an infection from the lung abscess. February 3, a tube was inserted in the eighth intercostal space in the posterior axillary line; 340 c.c. of foul putrefactive pus escaped. February 17, a subcutaneous abscess of the back was incised near the junction of the twelth rib with the spine. This abscess apparently communicated with the original intercostal incision. The ninth rib was resected. A large cavity, apparently of the whole right side of the chest, was revealed with the lung crowded against the mediastinum. By March 12, the patient was feeling much better, had been up in a chair for more than a week, and the sputum on some days was as little as 75 c.c. By March 24, the cough and sputum had increased, and it was seen that the patient was making no real progress.

It was decided to attack the abscess of the lung directly. The sixth, seventh, eighth and ninth ribs were resected for a distance of about 4 inches (10 cm.) each. A large portion of the exposed lung was burned out. The next day, she had practically no cough or sputum, but March 26, her cough had increased. The leukocytes were 33,000, and the temperature was practically normal. The wound was dressed daily with acriflavine, and, by April 20, there had been marked improvement. Sputum was now 75 c.c. daily. April 24, she had an attack of acute pain in the right upper quadrant of the abdomen suggestive of acute cholecystitis. May 4, without any anesthetic, another extensive cauterization of the lung was performed. Several small pockets of pus were encountered. No pain was experienced. Toward the last of the operation, there was hemorrhage from a large vessel which was readily controlled by packing. June 11, another cauterization was carried out, again without anesthetic. By this time, practically all the right lower and middle lobes had been removed. June 13, she showed signs of a massive pneumonia of the other lung, and she died, June 14.

Permission for a necropsy was refused. In reviewing this case, it seems probable that too many cauterizations were performed without sufficient intervals between them. It would have been better, perhaps, to have postponed the last cauterization for three or four months.

Case 4.—W. L., a white man, aged 41, admitted, Aug. 18, 1923, had a tonsillectomy, under nitrous oxid and oxygen anesthesia, in May, 1922. He had spat up blood continuously since the operation, except for intervals of four or five weeks at a time. He had had some severe hemorrhages. He had been unable to work, since October, 1922. There was dull, intermittent pain in the base of the right lung. There was sometimes foul sputum. The cough always was productive. There was about a pint (473 c.c.) of sputum daily. During August, the patient had had an artificial pneumothorax induced twice elsewhere.

There was impaired resonance in the right scapular region, less so in the base. Hyperresonance was present throughout the left chest. Below the angle of the scapula on the right side, the breath sounds were distant and

harsh. There were many fine, crepitant râles. Anteriorly, there was impaired resonance and diminished breath sounds below the nipple on the right side. There was definite clubbing of the fingers. Fluoroscopy and roentgen-ray plates showed a definite irregular shadow, which appeared smaller posteriorly than anteriorly. This shadow was apparently connected with the hilum and extended a little beyond the mammary line. When the patient was turned slightly, the periphery of the lung was seen to be free of the shadow. The diagnosis was chronic posterior lung abscess. The Wassermann reaction was negative. The sputum was consistently negative for tubercle bacilli. The complement fixation for tuberculosis was negative.

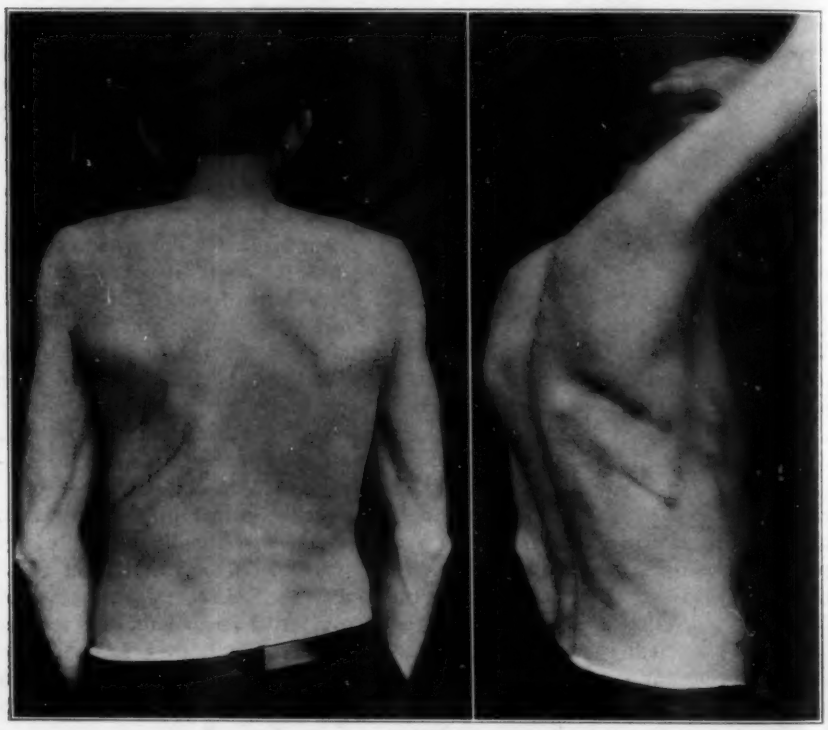

Fig. 2 (Case 4).—Healed wound after cautery pneumectomy; absence of deformity; same patient eight months after pneumectomy.

August 23, a pneumothorax was done by Dr. Singer; 300 c.c. of air was injected. August 24, 400 c.c. of air was injected. This was followed by much improvement and much less sputum. Postural drainage was also given every day. August 26, there was 50 c.c. of blood-streaked sputum. August 29, 650 c.c. of air was injected. Fluoroscopy immediately afterward showed the lung irregularly compressed with a suggestion of a fluid level in the dense mass. The patient was discharged, September 13, much improved. He reentered, September 27, because of the return of blood in the sputum. September 28, subperiosteal resection was done of 3 inches (7.6 cm.) of the eighth and the ninth ribs from the scapular line to the midaxillary line on the right side.

The lung was found adherent only in places, chiefly confined to the lower lobe. The pleura was sutured firmly to the lung. Light packing was done with iodoform gauze. October 8, without any anesthesia, a large crater was burned out of the lower lobe, and several small abscesses with thick pus were encountered. A fairly large artery began to bleed, but was easily controlled by packing. The patient suddenly refused to talk, and began to moan. It was impossible to explain this phenomenon; so the operation was terminated. In about ten minutes, he became rational again. December 7, he was discharged, having gained much in weight. His cough and sputum were practically gone. In the twenty-four hours, there was only enough sputum raised to cover the bottom of the cup. His vital capacity had increased from 3,900 to 4,100 c.c. His wound was healed, and the roentgen-ray examination showed the shadow in the lung to be much smaller.

Feb. 20, 1924, he was readmitted because of a sudden hemoptysis of about one-half teacupful of blood two days before. There was no more cough or sputum otherwise. February 23, a bronchoscopic examination was made by Dr. Arbuckle who discovered no source of the hemorrhage. In view, however, of the possibility of other hemorrhages, and with the belief that more compression might benefit him, the sixth and seventh ribs were also removed for a distance of 4 inches (10.1 cm.) from slightly posterior to the costal angles to the anterior axillary line. Bone wax was packed in the exposed ends of the ribs, and the adjacent intercostal nerves were injected with alcohol. It was felt inadvisable to cauterize further in view of our inability to locate the source of the hemorrhage. The patient was discharged, March 15. He is entirely well at the present time, and is at work. His wound is healed, and he has no more cough or sputum.

Case 5.—J. L., a woman, aged 21, admitted, Oct. 9, 1923, had been operated on elsewhere in March, 1922, for a perforated appendix. Three days later, she developed pneumonia and empyema, which left her in bed for four months. The empyema was drained, and the patient supposedly recovered with complete healing of the wound. In February, 1923, she caught a bad cold which did not improve. Expectoration became profuse, about a half teacupful daily. She entered another hospital, where she remained for sixteen weeks without improvement.

The patient was undernourished. There was marked clubbing of the fingers. There was an operative scar on the right posterior chest. Physical and roentgen-ray examinations showed what was considered to be an abscess of the middle lobe of the right lung connected with the hilum. The sputum was consistently negative for tubercle bacilli.

An artificial pneumothorax was tried, but was not effective. Nov. 2, 1923, the seventh, eighth and ninth right ribs were removed for a distance of about 4 inches (10.1 cm.), under nitrous oxid and oxygen anesthesia. The lung was found adherent in this region. Aspiration with the needle revealed some thick pus. With the cautery, a large opening was made into the lung, and a cavity was found containing about 20 c.c. of thick pus. In the cavity was also found a sequestrum of rib, about one-half inch long, which was removed. The abscess cavity was burned out with the cautery. The postoperative course was uneventful. For a few days, her sputum contained a little blood. She gained weight rapidly, and her cough and sputum disappeared. She was discharged, Dec. 17, 1923, with the wound healed, and she has had no further pulmonary symptoms since.

Case 6.—W. C., a man, aged 43, admitted, Aug. 10, 1923, had had a tonsillectomy elsewhere, Dec. 2, 1922, with ether anesthesia. About a week later,

he had a chill, and coughed up about a pint (473 c.c.) of greenish bloody pus. From then on to about March 1, 1923, this was repeated every third or fourth day. The weight fell from 147 to 117 pounds (66.7 kg. to 53.1 kg.).

The patient was emaciated. There was moderate clubbing of the fingers. The chest was flattened in the lower left base. There was dulness, bordering on flatness, with bronchial breathing in this region. Roentgen-ray examination showed an immobile left side of the diaphragm whose shadow merged with an irregular shadow in the lower left lung. The diagnosis was lung abscess and bronchiectasis. There was 500 c.c. of sputum daily. No tubercle bacilli were found.

There was steady improvement for three weeks with postural drainage and heliotherapy. The patient gained 12 pounds (5.4 kg.). The sputum was less. The patient was discharged, Sept. 6, 1923. He reentered, October 8, one month later, because during the preceding week he had been raising one-half teacupful of bloody purulent sputum daily. He had continued postural drainage at home. October 17, subperiosteal resection was done of the seventh, eighth and ninth ribs for a distance of 3 inches (7.6 cm.), with nitrous oxid and oxygen anesthesia. The lung was found adherent. No pus was found with the needle. A large cavity was burned out with the cautery. When the bronchi were opened, much fibrous tissue was encountered but no actual pus. The patient was in excellent condition when the operation ended. He came out of the anesthesia and talked to the anesthetist and assistants. Then, suddenly, he had a general convulsion with opisthotonus. The convulsion continued for about 30 seconds. He did not regain consciousness, and died ten minutes later in spite of artificial respiration and the injection of epinephrin into the heart. There had been no coughing and nothing to indicate much blood in the bronchial tree. It was considered to be a case of air embolism of the brain.

A necropsy failed to explain the death. There was marked edema of the cerebral pia-arachnoid. There also was extensive bronchiectasis of the left lower lobe.

CASE 7.—I. H., a girl, aged 17 years, admitted, Aug. 14, 1923, had begun in July, 1922, one month after a tonsillectomy with ether, to have pain in the left shoulder. She began to cough and to raise a moderate amount of foul yellow sputum at this time. She was given twenty injections of tuberculin by her local physician, although tubercle bacilli were never found. About two months after the tonsillectomy, she began having pulmonary hemorrhages. In March, 1923, she had a profuse hemorrhage, which was associated with the expectoration of a large amount of foul yellow sputum.

Physical and roentgen-ray examinations revealed evidence of abscess of the peripheral portion of the upper part of the left lung. Bronchoscopy (by Dr. Arbuckle) showed thickening of the mucosa of the larynx and trachea, which were covered with pus exuding from the left primary bronchus. Leukocytes were 31,000. The complement fixation test for tuberculosis was negative. The sputum was negative for tubercle bacilli. The Wassermann reaction was negative. There was moderate fever for the first five days in the hospital. This disappeared with rest in bed and postural drainage. Pneumothorax was considered not advisable because of the peripheral location of the abscess.

September 8, about 2 inches (5 cm.) of the fourth and fifth left ribs were resected through an incision parallel with the ribs from the anterior border of the scapula to the anterior axillary line. The lung was adherent to the pleura. Creamy pus was found on aspiration. The abscess cavity was entered with the cautery, and a large cavity burned out of the lung. There

Fig. 3 (Case 7).—Wound healed after cautery pneumectomy for chronic lung abscess; absence of any deformity.

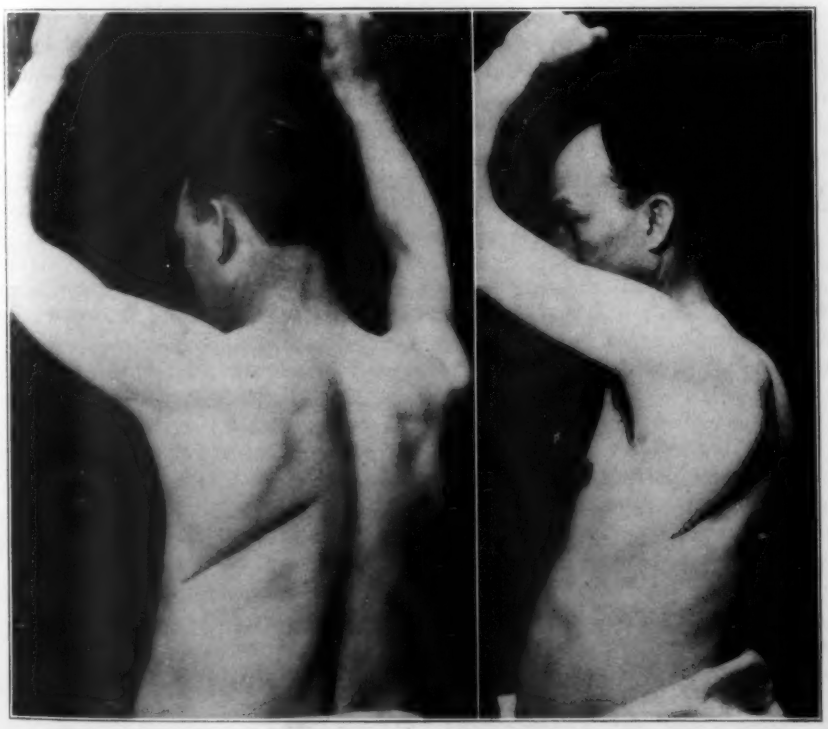

Fig. 4 (Case 8).—Complete healing of wound after cautery pneumectomy.

was no bleeding. The wound was packed open. The ends of the ribs were plugged with bone wax. The postoperative course was remarkably short and uneventful. The cough disappeared. There was a rapid gain in weight. The large bronchial fistula rapidly closed. The patient was discharged, October 4, and has remained perfectly well up to the present time.

Case 8.—E. H., a white man, aged 40, admitted to the medical service of Barnes Hospital, July 25, 1923, with the complaint of "cough," dated the onset of the present illness at Thanksgiving, 1922. For several months previous to this, however, the patient had been feeling bad, and had had pain in the chest. The weight, at that time, was 100 pounds (45.4 kg.). He also stated that for as many years as he could remember he had had a cough. He coughed night and day and raised a foul, dirty yellow sputum. The case was diagnosed pulmonary tuberculosis by the attending physician, and treated with serum and light rays. The patient consulted another physician, who told him that he had pneumonia with abscess formation. After seven weeks in bed, he was able to go back to work; but his cough persisted. He consulted a third physician, who advised an operation for drainage of the abscess.

The patient was fairly well nourished. The percussion note was impaired below the angle of the scapula on the left side. Breath sounds were impaired at the left base, and crepitant râles were present. Roentgen-ray examination showed, on the right side, an increase of hilum shadow and a thickening of lung markings in association with fine parenchymatous mottling. Confluent with the hilum shadow on the left was an irregular wedge shaped dense patch extending across the lateral chest wall. Within this area were indefinite dark areas suggestive of cavities. The blood Wassermann reaction was negative. The tuberculin complement fixation test was negative. The sputum culture yielded *Streptococcus viridans*. The dark field examination of the sputum was negative. No elastic tissue or acid fast organisms were found; phenolsulphonephthalein was 59 per cent. in two hours. Leukocytes were 14,000; the hemoglobin was 85 per cent. The urine contained a faint trace of albumin. The temperature rose to about 38.5 C. each evening.

With the patient under nitrous oxid ether anesthesia, July 30, 1923, the seventh and eighth left ribs were resected for a distance of about 4 inches (10 cm.). Intercostal bundles were excised. Intercostal nerves were injected with 95 per cent. alcohol. The pleura was not adherent to the lung, so it was not opened, but iodoform gauze was packed against it to produce adhesions. The wound was left open. One week later, the second stage of the operation was performed. The pleura was greatly thickened, and the lung was firmly adherent to the parietal pleura. Pus was not found with an aspirating needle even when one of very large caliber was used. The cautery was plunged into the indurated portion of the lung, and a cavity containing about 10 c.c. of very thick, almost caseous pus was found at a distance of about 2 cm. from the lung's periphery. The roof of the abscess was burned away. There was no hemorrhage. The cavity was lightly packed with gauze.

Following the operation, the patient improved rapidly. The cough became less; the drainage rapidly decreased, and the bronchial fistulas closed. The patient was discharged, August 26. One month later, fluoroscopy showed a faint shadow in the left axillary line which was thought to be thickened pleura. He has remained entirely well since that time. He weighs more than ever before, and he has no cough or sputum. He is steadily at work as an engineer.

Case 9.—F. G., a white man, aged 34, admitted to the medical service of Barnes Hospital, Sept. 13, 1923, with a complaint of "weakness and coughing,"

in March, 1919, had had influenza complicated by pneumonia on the left side. The patient did not recover fully. He lost 40 pounds (18.1 kg.) of weight and raised blood tinged sputum for the next three years. In April, 1923, the patient had a second attack of influenza with pneumonia. He was confined to bed for one month, and he had not recovered from this attack. He had coughed constantly and had had three severe pulmonary hemorrhages. The patient raised about one-third pint (157 c.c.) of sputum daily, most of which was expectorated in the mornings.

The patient was large and well nourished, with an offensive breath. There was dulness of the left base with absent fremitus and breath sounds, and many low pitched râles. Fluoroscopic examination showed a shadow above the left diaphragm. There was no fever. The blood Wassermann reaction was negative. The tuberculin complement fixation test was +. Leukocytes totaled 11,000; the hemoglobin was 85 per cent. Red blood cells were 4,900,000. The urine examination gave negative results. Phenolsulphonephthalein was 50 per cent. in two hours; nonprotein nitrogen 30.8 mg. per hundred cubic centimeters of blood. A sputum culture yieled pneumococcus Type IV; a dark field examination revealed no spirochetes. It was foul and blood-tinged. Elastic tissue was present; no acid fast organisms were demonstrated. The vital capacity was 3,200. A diagnosis of bronchiectasis of the left lower lobe was made, and it was thought that the case was a suitable one for lobectomy with cautery.

Postural drainage was successfully performed daily until the patient was operated on, Sept. 25, 1923. At the first stage of the operation, portions of the left seventh, eighth and ninth ribs were resected between the scapula and the axillary line. The lung was adherent only by delicate adhesions, so the pleura was not opened. The wound was packed with iodoform gauze. October 3, without anesthetic, a large crater was burned out of the left lower lobe with an actual cautery; enlarged, thickened bronchi were encountered which contained pus. October 17, more lung tissue was cauterized away through the old incision. November 5, without anesthesia, another portion of the lung was burned away. A brisk hemorrhage was encountered and controlled by packing with gauze. November 13, under gas anesthesia, the previous operative wound was extended backward below the scapula. The posterior ends of the ribs removed at a previous operation were resected for about 5 cm., exposing the lung posteriorly and medially to the region exposed previously. This portion of the lung was burned away with a cautery. Pockets of pus were encountered. The patient suddenly became pulseless and collapsed. There were no convulsions. A peculiar whistling sound occurred at the wound just before the collapse which made one think of air embolism. The wound was packed open and the patient was returned to the ward in a stupor. The patient recovered from the collapse. Fluoroscopic examination, December 6, showed that most of the left lower lobe had been removed. The patient was given heliotherapy. He was discharged, December 19, with only moderate improvement. Since then, however, he has become well, except that he has a small bronchial fistula which causes him almost no inconvenience. This result is probably partly due to the prolonged drainage in addition to the removal of a large part of the lower lobe.

Case 10.—A. F., a woman, aged 40, admitted, Jan. 7, 1924, had suffered the onset in January, 1918, with influenzal pneumonia and left otitis media. An operation was performed in February, 1918, for empyema. In November, 1918, the unhealed sinus of the empyema was injected with Beck's bismuth paste. Between November, 1920, and January, 1921, she had a thoracoplasty

for the obliteration of the pleural cavity and to remove the bismuth paste which was being extruded from time to time in small amounts. There was a repeated history of closure and opening of the fistula with the discharge of pus. Repeated examinations for tuberculosis were negative. The Wassermann reaction was negative. She still had cough and a moderate amount of sputum, with attacks of fever and pain in the chest.

Jan. 19, 1924, an incision was made around the old sinus on the right side at about the level of the seventh rib in the posterior axillary line. With a large probe in place, the sinus was dissected up and found to enter a cavity about 3 inches (7.6 cm.) above and posterior to the opening of the sinus, which had at the other end a bronchus. This seemed to be a definite bronchiectatic cavity. It was burned out with the cautery. The cavity contained about 40 c.c. of thick pus with a putrefactive odor. The patient was discharged, February 24, with an open bronchial fistula, and was greatly improved in all respects. There was no cough or sputum. Since then, on two or three occasions, the fistula has closed spontaneously, and she has had pain, cough, sputum and fever until it has reopened itself. She is, however, greatly improved. It is probable that a more extensive cauterization would have given her greater benefit. There is probably still some diseased lung remaining.

Case 11.—A. H. N., a white man, aged 37, who entered Barnes Hospital, Jan. 2, 1924, complaining of "lung abscess" had in the family and past history nothing related to the present illness. One year before (1923), the patient had a tonsillectomy under general anesthesia. One week later, he developed double pneumonia, which developed into a lung abscess. At present, the patient coughed frequently and raised approximately 18 ounces (532 c.c.) of sputum in twenty-four hours. He had lost 15 pounds (6.8 kg.) in weight in the last year. He had been treated by postural drainage without success. In June, 1923, the patient had a pneumothorax performed fifteen times. The lung was never successfully collapsed because of pleural adhesions. The present illness had confined the patient to his bed.

The general condition of the patient was good. There were physical signs of abscess of the left upper lobe of the lung. Roentgen-ray examination of the chest showed an area of the left lung from about the upper border of the third rib anteriorly to the apex to be almost totally opaque. This region was bounded below by a fairly well defined line of aerated lung. There was a striated infiltration connecting this area with the hilum. There was some thickening of the lung markings in the rest of the field. The right lung showed a dense hilum increased in size with much calcification. The blood Wassermann reaction was negative. No tubercle bacilli were found in the sputum.

A pneumothorax was made, introducing 500 c.c. of air and securing collapse of the lung everywhere except at the apex. January 7, a section of the third left rib anteriorly was resected for a distance of about 3 inches (7.6 cm.). The lung was free anteriorly but adherent posteriorly to the chest wall. The upper portion of the lobe was firmly indurated. The wound was closed, as it was felt that it would be necessary to approach the abscess posteriorly to avoid an empyema. January 16, an incision was made between the border of the scapula and the vertebral column with the patient, under nitrous oxid-oxygen anesthesia. Portions of the second, third and fourth ribs were removed subperiosteally. The pleura was incised; the lung was adherent. Exploration with a needle revealed a small amount of thick pus. The area was cauterized. Through the old incision, January 28, a still larger portion of the upper left lobe was burned away. The patient improved steadily, though some coughing

persisted. March 17, the lung was explored through the old incision. The lung was found to have become fibrotic. The aspirating needle was inserted into the lung in various directions, but no pus was found. The wound was left open. April 9, the patient developed signs and symptoms of a brain embolism. April 15, the patient gave evidence of a meningitis. A lumbar puncture was made. The fluid was cloudy. There were 15,000 cells per cubic millimeter; 98 per cent. of which were polymorphonuclears and 2 per cent. lymphocytes. The patient died the following day.

Case 12.—T. T., a white man, aged 24, who entered Barnes Hospital, Jan. 3, 1924, with the chief complaint of "cough and weakness," had a negative

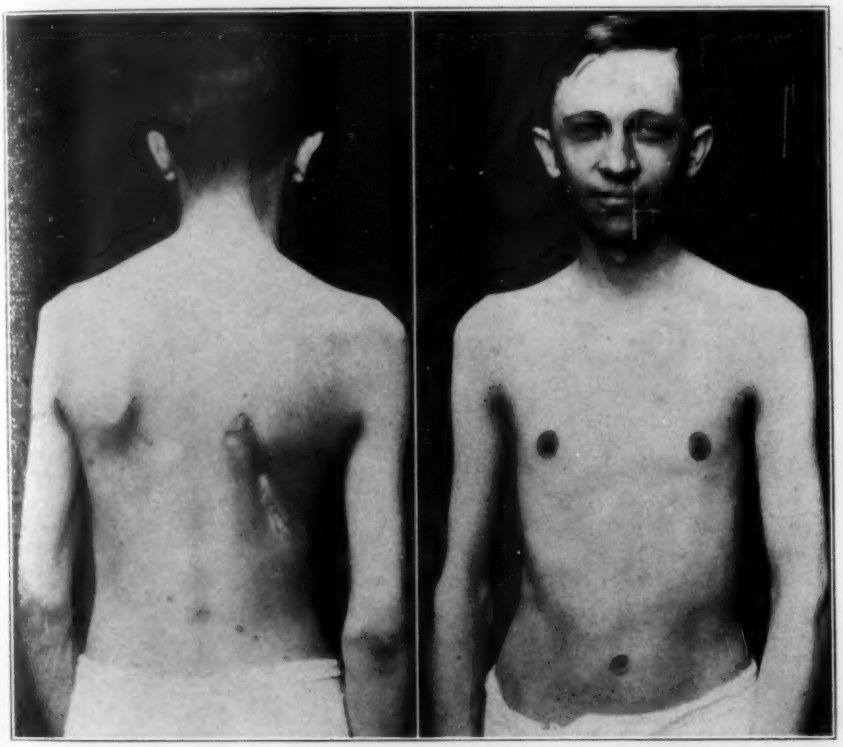

Fig. 5 (Case 12).—Cautery pneumectomy for bronchiectasis of many years' standing; patient has bronchial fistula, but is very much improved.

family history. He had had pneumonia when seven years of age. The patient was in good health until 12 years of age (1911). At that time, he fell off a bicycle, which caused him to raise two mouthfuls of thick yellow material. There was no cough or discomfort until another attack, one year later (1912), when he became suddenly ill with headache and chill. The patient coughed up a cupful of thin yellowish material. He soon felt better and gained weight. In 1915, he began to lose weight and feel weak. He began to cough up considerable sputum on retiring, but never in the morning. At this time, he went to the Pacific coast where he gained weight and stopped coughing. In 1917, he took a bad cold and began coughing in the morning. About two months

later, he raised a large amount of sputum and since that time had had a productive cough. The condition was diagnosed as pulmonary tuberculosis. Beginning in February, 1922, the patient stayed in bed for seventeen months on the advice of a physician. In July, 1923, his condition was diagnosed as lung abscess, and, July 28, portions of three ribs were removed, elsewhere, and the lung was cauterized and drained. After a stormy postoperative course, the patient gained weight and the cough decreased. Three weeks after the operation, the patient had a pulmonary hemorrhage. Since the chest wound

Fig. 6.—Drawing made from Case 12 before the cauterization was completed, illustrating the massive drainage permitted by the cauterization of a large area of lung surface. Many bronchioles are opened up and permitted to discharge their contents to the surface.

had closed, the patient coughed considerably and raised about three cupfuls of sputum daily.

The young man was emaciated. The mucous membranes were pale. Over the region of the right base of the lung, posteriorly, there was an old operative scar, and there was a local collapse of the chest wall. The percussion note was resonant everywhere except over the right base where it was dull from

the angle of the scapula downward. Over this region, the breath sounds were of the bronchial type, and whispered sounds were increased. Râles were heard in both bases. A culture of sputum showed *Streptococcus viridans.* An examination of the sputum for spirochetes and acid-fast bacilli was negative. The blood Wassermann reaction was negative. The urine examination gave negative results. Phenolsulphonephthalein totaled 50 per cent. in two hours. There was no anemia. The roentgen-ray examination showed that the ninth, tenth and eleventh ribs on the right side had been resected about the midscapula line. Beneath this area was a large area of the density of the lungs, confluent with that of the heart. There was a slight increase of the hilum shadow with fairly extensive thickening of the lung markings. The patient did not have fever. From the findings, it was concluded that the patient was suffering from a bronchiectasis of the right lower lobe of the lung with abscess formation.

Jan. 12, 1924, under local anesthesia, an incision was made through the old scar. Two of the regenerated ribs were removed. The lung was firmly adherent to the chest wall. With the cautery, a large cup shaped area was burned away from the lung. Several pockets of pus were entered and destroyed. A small but brisk hemorrhage was encountered which was easily controlled by packing. When the packing was removed, three days later, the patient lost about 200 c.c. of blood from an intercostal artery. But he improved considerably at once. Nine days later, as a preparation for further operative procedures, the patient was transfused with 500 c.c. of blood. January 29, more lung tissue was burned away with a cautery. Vigorous bleeding was encountered, but it was controlled by ligation of the bleeding vessel. The cautery was passed upward close to the spine to enter a fairly large bronchiectatic cavity containing thick pus. February 21, the old incision was opened, and a considerable amount of lung tissue containing pus coming from greatly thickened and enlarged bronchi was removed with the cautery. A rather severe hemorrhage occurred, but this was easily controlled by packing. As the wound began to close, the patient coughed more. April 30, the wound was reopened to permit freer drainage. No cauterization was done. The patient left the hospital, May 14. He was observed, until June 25, when he was allowed to return to his home in New Mexico. A letter from the patient, July 15, stated that he continued to cough some and had some drainage from his chest but had gained weight and strength, being able to drive an automobile and to go fishing and hunting. He was to return in September for examination.*

This patient was practically bedridden for the two years before his admission. His improvement was gratifying.

Case 13.—B. D. J., a white man, aged 41, referred by Dr. W. B. Ruess, San Antonio, Texas, entered Barnes Hospital, Feb. 14, 1923, with the chief complaint of lung abscess. The past history was irrelevant to the present complaint. July 9, 1922, the patient had a tonsillectomy performed under general anesthesia. He seemed to be recovering satisfactorily until ten days after the operation,

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* On his return for examination, he stated that he considered himself as practically well. He had gained 22 pounds (10 kg.), and he was able to do anything he wished without abnormal fatigue. His wound had three unhealed bronchial fistulas from which a small amount of mucopus was discharged. He had an occasional cough with the expectoration of about 50 c.c. of sputum daily which probably came from an involvement of the middle lobe which had not been operated on. He was sent home again for a period of a year to decide whether more cauterization would be necessary.

when he developed fever and had to go to bed. A few days later, the patient began to cough and raise a large quantity of seropurulent sputum. The middle of August, 1922, the patient went to the mountains of Texas where he gained weight and felt better, and the amount of sputum he raised decreased to about one cup a day. He returned home, and his condition rapidly became worse. In December, 1922, drainage of the lung abscess was established through the chest wall after the resection of some ribs. Improvement followed the operation until the wound began to close, causing expectoration of more sputum and the recurrence of fever. In May, 1923, the patient returned to the mountains, his chest wound nearly closed, but he did not find relief there. Postural drainage was tried without improvement. The patient had been in bed since September, 1923. He coughed frequently and raised from 600 to 800 c.c. of purulent sputum daily.

Physical examination of the patient showed him to be fairly well nourished. There was a 3 inch (7.6 cm.) diagonal scar over the right upper chest posteriorly. In this region, the breath sounds were diminished, and there was dulness on percussion. Roentgen-ray examination showed an area in the right upper posterior portion of the chest of almost total opacity joined to the hilum by a striated band. There was an increase of hilum shadow bilaterally, and a thickening of lung markings. When viewed with the fluoroscope, the area seemed to be divided into two cavities which did not communicate. The patient had a low afternoon fever. The blood pressure was 75 systolic, 45 diastolic. The blood Wassermann reaction was negative. The urine examination gave negative results. There was no leukocytosis or anemia. The sputum was negative for tubercle bacilli. This case of lung abscess did not seem a suitable one for pneumothorax, but rather one for cautery pneumectomy.

An operation was performed, Feb. 16, 1924, under nitrous oxid-oxygen anesthesia. The incision was made through the old scar. A plate of regenerated bone was removed with difficulty. A multilocular abscess cavity was found which contained altogether about 2 ounces (59 c.c.) of thick foul pus. The overlying lung tissue was completely burned away with a cautery leaving all parts of the cavities widely exposed. The cavity was packed with petrolatum gauze. The patient's general condition was improved following the operation, but he continued to cough and raise considerable sputum. It was thought advisable to remove more lung tissue and establish better drainage. April 9, the chest wound was reopened. A large amount of fibrous tissue was found everywhere in the wound. Soon after cauterization was begun, the patient inhaled a small quantity of blood which caused a violent coughing spell of about ten minutes' duration. It was decided to postpone further operative procedures. Heliotherapy to the abscess cavity was instituted. April 26, a cup shaped portion of the lung about the abscess cavity was burned away until the normal lung tissue was met with all the way around. There was no hemorrhage. The patient did not recover promptly from the gas anesthesia, however; he remained unconscious and coughed frequently. There was a right facial weakness. The legs were spastic. The patellar reflex was exaggerated. There was marked ankle and patellar clonus. The optic disk did not show emboli. A neurologic consultation was held with Dr. S. Schwab, who was of the opinion that the condition was due to a cerebral embolism. The patient began to have convulsive seizures of the right side of the body the following day. He became worse and died. No necropsy was done. Except for the accident of cerebral embolism, there was every reason to expect an excellent result in this case.

CASE 14.—C. P., a white girl, aged 11 years, who entered St. Louis Children's Hospital, Jan. 5, 1923, was an orphan who had been under the care of a foster-mother for six years. The onset was in December, 1921, with cough and a mild cold which cleared up. In January, 1922, the patient had pain in the right side with fever and cough, and pneumonia was diagnosed. In February, empyema was diagnosed. She was operated on in May, 1922, elsewhere, for empyema under the right scapula. Drainage was continued until September. Since then, she has drained only a very little through the small sinus, and she has had cough, sputum and fever. She has had three pulmonary hemorrhages. The sputum has a foul odor.

Examination showed a small sinus discharging pus, immediately below the angle of the right scapula. There was marked scoliosis, emaciation and clubbing of the fingers. The sputum was negative for tubercle bacilli but contained many

Fig. 7 (Case 14).—Wound entirely healed except for small bronchial fistula; the patient is free from symptoms for the first time in more than two years; there is still some scoliosis present, but much less than when the patient was admitted.

staphylococci. The complement fixation test for tuberculosis was negative. The Wassermann reaction was negative. The temperature was 101 F.

January 15, the seventh and eighth ribs immediately above the sinus were resected for a distance of about 2 inches (5 cm.) with nitrous oxid and oxygen anesthesia. The opening into the pleural cavity was enlarged, and a large amount of old fibrin and thick, putrid, inspissated pus was found. Two large drainage tubes were inserted, each containing a Carrel tube for irrigation. A piece of pleura examined microscopically showed no evidence of tuberculosis. Irrigations could not be carried out because the fluid ran into the lung in all positions in which the child was placed. Marked improvement was shown following this operation with a gain of 5 pounds (2.3 kg.) in weight. The patient was discharged, June 23. She was readmitted, October 1, for cautery

pneumectomy because of the persistence of cough, fever and sputum, although she had gained 6 pounds (2.7 kg.) more, making a total of 11 pounds (5 kg.) since her operation, January 15. The pleural cavity had become obliterated, leaving the lung adherent to the chest wall with the sinus communicating directly with an open bronchus. October 3, the old sinus was dissected out, and four ribs which had become rotated and were so close together that they were touching were removed. The ribs removed were apparently the eighth, seventh, sixth and fifth. The sinus was found to lead laterally and anteriorly into a large cavity which was situated within the lung itself. This cavity was filled with old inspissated pus with a very foul odor. There were also several large bronchial fistulas opening into the large cavity whose capacity was estimated at 100 c.c. The roof of the cavity was everywhere removed with the cautery, leaving a cup-shaped depression in the lung which communicated directly with the external wound. The convalescence was uneventful. The child was now perfectly well, except for a bronchial fistula from which there was practically no discharge. Cough and sputum were absent.

Case 15.—A. K., a white boy, aged 10 years, who entered St. Louis Children's Hospital, Nov. 14, 1923, suffered the onset with influenza in 1918, since which time cough and sputum have always been present. He coughed up 250 c.c. of pus during his examination.

Examination showed signs of an abscess in the left lower lobe, probably with bronchiectasis.

Postural drainage was instituted for about three weeks with only slight improvement. December 27, the eighth and ninth left ribs were resected from the posterior axillary line to just beyond the angles of the ribs. The lung was found to be nonadherent. The lower lobe was sutured to the parietal pleura. The wound was packed open with iodoform gauze. Jan. 9, 1924, a large cup shaped portion of the lower lobe was burned out. It was estimated that about one-half the lobe was removed in this way. No bleeding occurred. January 29, additional cauterization was carried out. No bleeding occurred. Uneventful convalescence followed, except for the development of an abscess of the jaw about two weeks after the patient's discharge, March 3. At the present time, he is entirely well for the first time in six years, except for a small bronchial fistula from which a little mucus escapes.

Case 16.—D. B., a colored boy, aged 5 years, admitted, Sept. 6, 1923, had pneumonia in August, 1922. Thoracostomy was performed at the City Hospital in September. The chest was drained for seven weeks, and healed. In March, 1923, the patient had measles, and, since then, had had fever. An abscess in the region of the right scapula ruptured. The patient coughed frequently and raised considerable foul sputum.

Dulness was present below the fourth rib anteriorly and posteriorly. Breath sounds were absent. With the fluoroscope, there could be seen a dense shadow below the fourth right rib, annular in shape.

Under ether anesthesia, September 8, a 3 inch (7.6 cm.) incision was made from the right posterior to the anterior axillary line. The lower lobe was indurated and felt nodular. The lobe was adherent to the pleura. Almost the entire lower lobe was removed with an actual cautery. During this process, six different abscess cavities were found. The lobe resembled a sea sponge saturated with pus. Near the conclusion of the operation, a large vessel was cut through with the cautery, and brisk bleeding occurred for a moment. This

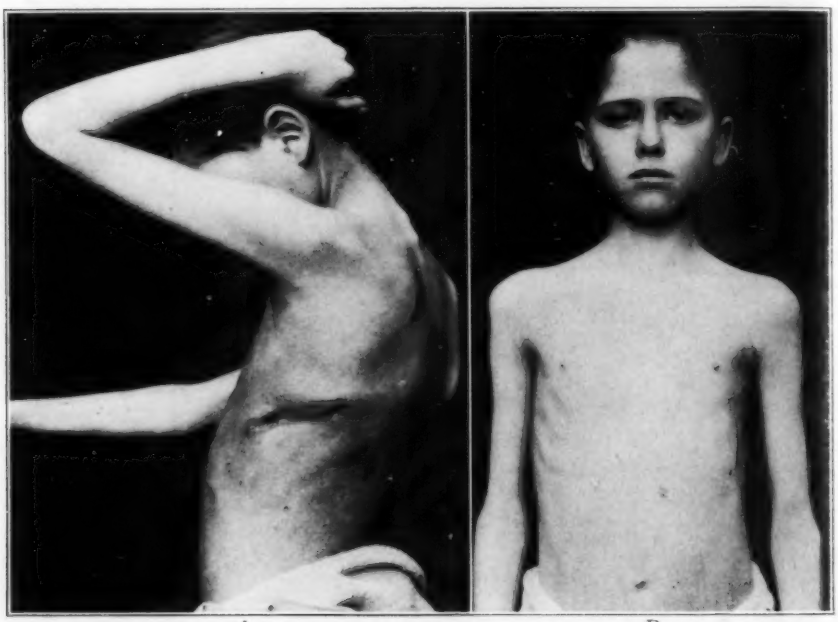
was readily controlled by packing. The patient improved rapidly for a month, when he developed a bronchopneumonia from which he recovered. He was discharged from the hospital, December 23, with his wound entirely healed except for the opening of a bronchial fistula. He is being observed in the outpatient department. His general condition is good, and he is free from symptoms. The fistula has not closed as yet.

Fig. 8 (Case 16).—Patient free from symptoms and well except for small bronchial fistula after cautery pneumectomy.

CASE 17.—G. M., a white boy, aged 10 years, was admitted to the St. Louis Children's Hospital, March 3, 1924, with complaints of pain in the stomach, afternoon fever, vomiting, cough, expectoration and loss of weight. A maternal aunt had died of tuberculosis three years before. One month previous to admission, the patient became sick with fever, cough and malaise. He returned

to school after remaining in bed for three days, but one week later he became ill again with abdominal pain and headache. He vomited frequently. Since his first illness one month before, he had coughed frequently and raised yellow sputum.

The patient was poorly nourished; the temperature was normal. The percussion note was dull to flat in the left axillary space and anteriorly to the third rib. Breath sounds were increased over this area. Numerous râles were present. The fingers were clubbed. With the patient under a fluoroscope, a dense circular shadow about 2½ inches (6.2 cm.) in diameter was visible in the region of the fourth, fifth and sixth left ribs. The blood Wassermann reaction was negative. The pirquet reaction was negative. The results of the urine examination were negative. No leukocytosis was present.



A B

Fig. 9 (Case 17).—*A*, seven weeks after operation of cautery pneumectomy; patient is free from symptoms and entirely well except for small bronchial fistula; *B*, there was no scoliosis and no deformity six weeks after the operation.

March 10, operation was done, under nitrous oxid-oxygen anesthesia. About 2 inches (5 cm.) of the seventh and eighth ribs were resected in the mid-axillary line. The lung was adherent above the level of the eighth rib. An abscess was found with the aspirating needle. The cavity was opened with actual cautery, and a large cup shaped area was burned away. About 8 c.c. of thick inspissated pus was found in the abscess cavity. A small soft rubber tube was put into the main bronchus leading into the cavity to prevent subsequent closure from edema. Ends of the ribs were plugged with wax. The patient was discharged, April 23, after an uneventful postoperative course. On discharge there was no cough, the general condition was good; only a small sinus remained.

CASE 18.—J. L., a man, aged 52, admitted to Barnes Hospital, Oct. 26, 1923, had coughed since he was 17 years of age. He had pleurisy, two years before admission, on the right side, and had four other attacks since then. He had gonorrhea thirty-five years before; chancre, thirty-four years before. He had had mercury and arsphenamin. He had been told that he had diabetes two weeks before. For the last two years, he had been bringing up a large amount of sputum.

Examination revealed moderately severe diabetes. The Wassermann reaction was four plus. The chest examination revealed a hydropneumothorax or a pyopneumothorax. Aspiration in the ninth interspace withdrew 230 c.c. of cloudy greenish yellow fluid.

This fluid gradually became purulent, and Nov. 14, a thoracostomy was performed with resection of the ninth rib, and 1,000 c.c. of thick, putrid, creamy pus was removed. A large bronchial fistula leading into the lower lobe was noted. December 15, because of the persistency of cough and sputum, a needle was inserted into the lung in various directions, but no pus was found. The indurated lung around the old bronchial fistula was then extensively cauterized. Two days later, tubercle bacilli were found in the sputum for the first time and, later, were found on several occasions. His convalescence has been slow but steady. He now weighs 20 pounds (9 kg.) more than his lowest weight while in the hospital. There is only a little cough and practically no sputum. He occasionally has fever of 100 F. He still has a large unobliterated cavity in his pleural space, and he still has a bronchial fistula. The result, in this case, seems particularly encouraging in view of the fact that he had a combination of many serious conditions, diabetes, syphilis, tuberculosis, empyema and a lung abscess which was probably due to a mixed infection of a pyogenic organism and tubercle bacilli.

CASE 19.—G. V. B., a man, aged 41, admitted to Barnes Hospital, March 3, 1924, had a gastro-enterostomy, elsewhere, for gastric ulcer in September, 1920. Nine days later he developed a pneumonia of the right lower lobe and was acutely ill for one month. He continued to cough and to expectorate. In the spring of 1921, he began to expectorate blood. He was told that he had an unresolved pneumonia; then he had a diagnosis of tuberculosis made, although bacilli were not found in the sputum. He went to a tuberculosis sanatorium in the west, where he had six severe pulmonary hemorrhages. He was in the sanitarium for seven months, and his condition was diagnosed as lung abscess. Postural and bronchoscopic drainage were carried out. He had bismuth injected through a bronchoscope. He returned home in June, 1922, and remained there for one year. From July to November, 1923, he had seven thoracoplasty operations done elsewhere. There was no improvement in any respect.

Examination showed the excellent technical result of a right-sided thoracoplasty. Roentgen-ray examination showed numerous foreign bodies in the bronchial tree (bismuth?). There was opacity of the whole right lung, most dense in the lower lobe. In the upright position, there could be seen a small region of air-containing lung in the axillary, and also in the mediastinal regions. A diagnosis was made of bronchiectasis, chiefly of the lower lobe. Hemoglobin was 60 per cent. Leukocytes totaled 8,150. The urine was normal. The sputum showed no tubercle bacilli and no spirochetes. It measured from 300 to 500 c.c. daily. The temperature was normal. The Wassermann reaction was negative.

March 29, a curved incision was made along the line about where the eighth rib would be normally. Some regeneration of the ribs was found, so that the chest wall was rigid and not collapsed. New bone was removed. Soft tissues

were dissected up. The lung was freely exposed, and the lower lobe, which was adherent to the pleura, was found to be cyanotic and indurated. With pressure, coughing was induced. The sputum contained pus and a little blood. None of the masses of bismuth seen in the roentgenogram could be felt. An aspirating needle was inserted in several directions, but no pus was found. With a cautery, a large area was burned in the lower lobe, and, in several places, a few drops of pus were obtained from the open bronchi. April 8, without anesthesia, more cauterization was carried out. Several large bronchi were cut across with the cautery. No pain and no marked bleeding resulted. May 3, the old incision opened up widely. Several fragments of newly formed rib were removed with a cautery, and more lung tissue was burned out. Many large bronchi opened up widely. May 4, the patient slept well. There had been no cough or sputum since the operation. Blood transfusion was done because of low hemoglobin; 500 c.c. were given. Hemoglobin was 55 per cent. There was no hemorrhage from the lung. June 21, the incision was enlarged. A small cautery was plunged into the lung at various places for a distance of about 1 to 1½ inches (2.5 to 3.7 cm.). A cavity about the size of a hen's egg was burned out with a cautery, and then a large blood vessel was encountered which gave very vigorous bleeding. This hemorrhage was easily controlled, but it prevented further cauterization of the lung.

July 1, sputum and cough were only about one half of what they were at the time of the patient's admission. He stated that he felt better than at any time since the beginning of illness. He was discharged, July 22, to return in September or October.

As subsidiary treatment, he was given heliotherapy and aspiration of the open bronchi. This case is still in progress. More cauterization will be done.

Case 20.—T. R., a white man, aged 35, had a lung abscess of the right lower lobe of eight months' duration which occurred immediately following an operation on the gallbladder done elsewhere. Treatment is still in progress, and the patient is still in the hospital. Following two extensive cauterizations of the affected lobe, the patient was very much improved, and the cough and sputum greatly diminished. It is probable, however that he will need further cauterization.

AN INSTRUCTIVE CASE OF ABSCESS OF THE LUNG ASSOCIATED WITH MEDIASTINAL TUMOR

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NEW YORK

M. C., an Italian woman, aged 45, who had lived fourteen years in New York, beyond the facts that three of her children had died in infancy (three were living and well), that her menopause had occurred at 38, that she had had right sided supra-orbital headache for nearly ten years, and that she suffered somewhat from constipation and "gas," had always been strong and well until thirty-six days before admission to the hospital, March 8, 1923.

She awoke one morning with a severe headache, but worked at household duties for a day or two until a cold in the head, then fever, then cough and then greenish yellow sputum combined to force her reluctantly to bed. The physicians thought that she had influenza and bronchopneumonia, and sent her to the hospital because she was getting worse and they suspected that she had an empyema.

Certain features in her physical examination predominated. She was manifestly tired from incessantly coughing large amounts of greenish, purulent sputum. The odor was not offensive. She looked pale, and her count showed a moderate degree of a secondary anemia. Red blood cells were 3,300,000; hemoglobin, 75 per cent. There was dyspnea, fever and leukocytosis; white blood cells were 19,000; polymorphonuclears, 70 per cent. No tubercle bacilli could be found. The heart was not displaced. The Wassermann reaction was negative. The signs of a diffuse bronchitis with many moist râles were readily made out over both chests posteriorly, but the main signs were at the left base in the posterior axillary line. Here were dulness and moist râles, but diminished voice, breath sounds and fremitus. There was no coin sound or splash, nor even a suggestion of cavernous or bronchial quality to the sounds. Roentgen-ray examination showed a small cavity containing air with apparent fluid level, so that a diagnosis of a small empyema with pleuropulmonary fistula was considered most probable, and operation was done.

The first feature of peculiar interest then appeared: Rib resection was done under local anesthesia, and a small empyema cavity containing air and about 50 c.c. of greenish white pus opened widely. Free, in this cavity, lay a disk of dead lung about the size of a silver dollar. It had completely sequestrated from the lung surface, leaving a round, shallow, bluish concavity with two bronchial openings, an area sharply demarcated from, and in striking contrast to, the visceral pleural surface surrounding it. When this bit of lung sequestrum was picked up with forceps and placed in the concavity, it was a precise fit. Sections of this sequestrum showed, in ghostlike form, the lung architecture, alveoli, trabeculae, thrombosed vessels, bronchi and pleural surface, with cells that had lost their outline, their nuclei and staining properties, in contrast to many leukocytes and pus cells on its surface that stained well. Here, then, was found lung parenchyma forming part of the wall of a cavity containing air and pus. It might, with considerable plausibility, have been called a lung abscess, and if it had been seen at a late stage might have resembled

Fig. 1.—Lung sequestrum free in empyema cavity, removed at operation.

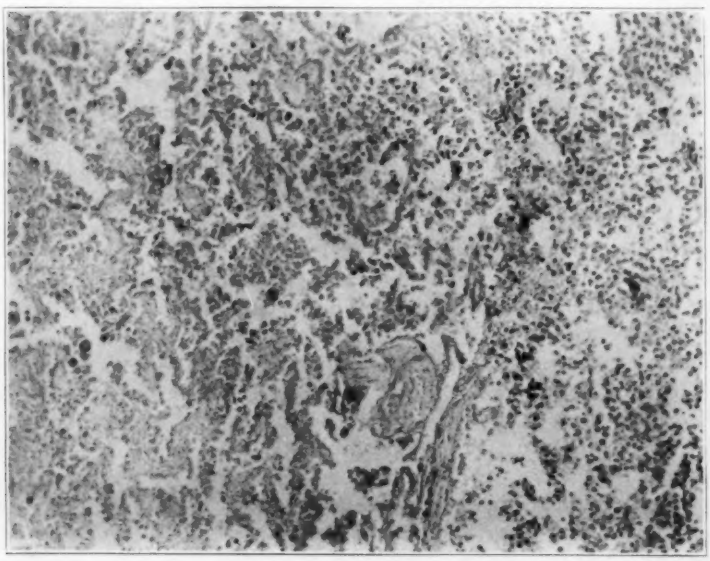

Fig. 2.—Section of lung sequestrum; only cells that stain well are the leukocytes; most of it made up of the remains of alveoli.

Fig. 3.—Lung sequestrum, showing lung architecture but no normal cell structure nor cell staining.

one still more. But the sequestrated lung showed no evidence of a lesion that began with central necrosis and progressed with peripheral spread. Rather had death of lung tissue occurred suddenly and en masse, as if an infarct had occurred from thrombosis of the vessel supplying the sequestrated tissue. Such a happening in the presence of infection best accounts for this picture. Culture of the pus grew out a hemolytic streptococcus and a Pfeiffer influenza bacillus. Nor is this just the picture of a so-called "subcortical abscess" so beautifully described by others, resembling it as it does.

The second feature of interest in this case lay in observations made during the succeeding few weeks as to the changes that occurred in the healing of such a lung lesion. A small empyema cavity and much smaller lung cavity

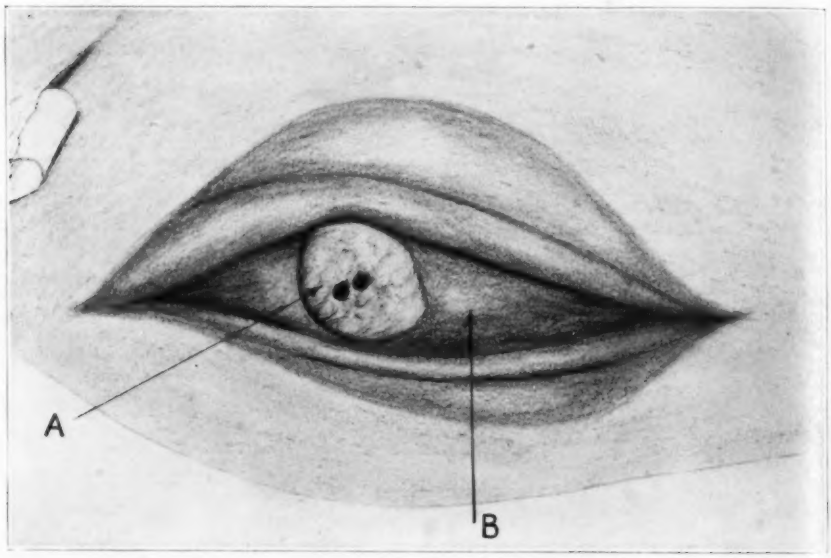

Fig. 4.—Wound, March 18, 1922, six days after operation; blue lung parenchyma with conspicuous absence of red granulations appears as a shallow concavity. This cavity corresponds precisely to the disk of dead sequestrated lung found free in the empyema cavity. *A*, two bronchial openings; three days later, there were three. On deep inspiration, the blue parenchyma retracts as the chest wall goes out; the bronchial openings remain open but become oval with the long axis extending up and down as though diaphragm excursion were an important factor, and the whole cavity becomes less shallow and more cone shaped. When the abdominal and chest walls contract and exert pressure on the lung against a closed glottis in coughing, the blue parenchyma bulges so as to bring the surface flush with the visceral pleura, *B*, and compresses the bronchial openings so that they quite disappear. A small amount of exudate or secretion mixed with air bubbles comes out of these openings just before they close off completely.

remained in open view for study. The pleural surfaces, the muscles and subcutaneous tissue, in a few days, were, of course, coated by a smooth lining of red granulations. Not so the lung parenchyma. This could not be said to "heal by granulation." The dominant color of this tissue remained blue,

though, as time went on, whiter trabeculae appeared running across the field and about the bronchial openings. These trabeculae became pink and more vascular when the patient strained or coughed. The number of bronchial openings increased instead of diminishing. Whereas only two were at first present, there were three in a few days, then five and, finally, seven. Now, later it was ascertained at necropsy that these bronchial openings were the terminal branches in direct line with the main branches to that lobe. For more bronchial openings to appear in this manner, it would seem that the delicate alveoli between them must have disappeared through atelectasis or destruction. The shallow depression found at operation deepened. Its central portion retracted into the chest so that it became cone shaped. Its opening on the surface became narrowed through the throttling action of a dense ring of scar tissue. The empyema cavity became quite obliterated, and the ring of scar tissue deepened so as to be composed of granulation tissue from not only the fused pleural

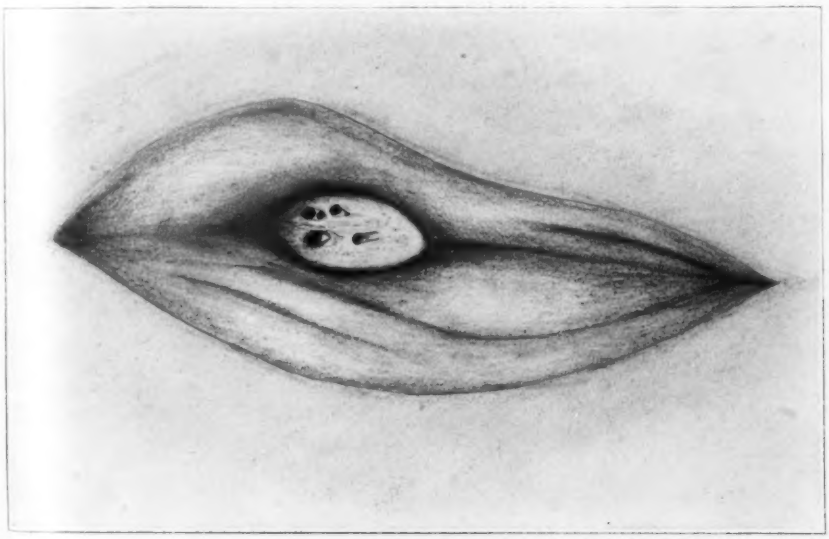

Fig. 5.—Wound, April 9, twenty-eight days after operation; empyema cavity has practically gone. The lung parenchyma is withdrawn deeper into the chest. Its diameter is lessened. A dense ring of scar tissue throttles the sinus leading to it. Five bronchial openings are present instead of two. There is not as much blue lung. There are white strands and trabeculae which stand out in relief as the blue parenchyma is sucked in during the inspiratory effort. When the patient coughs a few times, these white strands become pinker as though congested with aerated blood.

surfaces but also the remaining tissues of the chest wall. The purse string action of this ring of contracting scar tissue converted the originally wide open shallow concavity into a sinus, so that the retracting walls of the lung cavity were brought closer and closer together, and eventually scarcely more than a slit could be seen. Certain other features could be readily seen. In deep inspiration, the effect of the descent of the diaphragm was such as to pull the adherent lung well down, turning the bronchial openings into an oval

instead of normally round shape. So great was the pressure exerted by the contraction of the chest and abdominal muscles against a closed glottis on coughing, that the air-containing lung was made to bulge, actually closing off the visible bronchial openings so that they could no longer be seen. A small amount of fluid with air bubbles was squeezed out of them just before they were closed off entirely.

A third feature of interest lay in the interpretation of shadows in several roentgenograms taken at intervals during the thirteen weeks following operation. Just before operation, a cavity shadow at the left base showing air and a horizontal fluid level made the diagnosis of pyopneumothorax pretty evident. A shadow to the right of the midline, however, thought to be evidence of a diffuse dilatation of the ascending aorta, showed plainly, as did also a less prominent concavity high on the left side, that was assumed to be the beginning of the descending portion. These shadows, modified, it is true, by

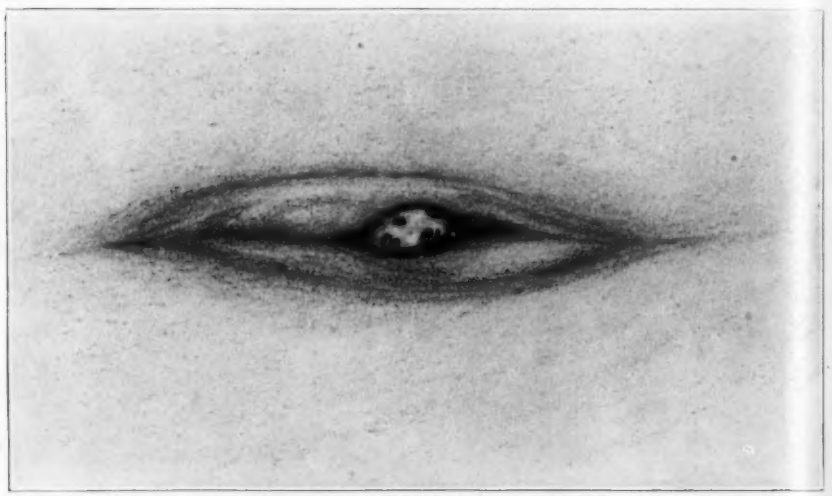

Fig. 6.—Wound, April 24, forty-three days after operation; the whole wound much smaller. The scar tissue ring has throttled the diameter of the exposed lung area to about one-quarter its former size. There are seven bronchial openings that become markedly ovoid on deep inspiration. There is less blue tissue and more white trabeculae, which become reddened when the patient strains or holds her breath in any effort. Occasionally, peculiar motions occur not synchronous with respiration, slow contractions with shrinkage and expansion changes, suggesting a sort of peristalsis. It would seem that an atalectatic process of considerable degree had taken place, as evidenced by the lessening of the finer structures of the lobules and increase in evidence of bronchi and connective tissue trabeculae. No granulations cover this lung surface. It appears to be receding within the chest while a cicatrizing ring of scar tissue gradually narrows the opening as would a purse string.

the varying subsequent conditions, were evident in all the later plates. Their significance was not clear until necropsy.

The patient did well except for a persistently low red cell count for which she was transfused; she was up and about until the seventeenth day, when she

had an elevation of temperature as high as 104 F., leukocytosis and signs of a bronchopneumonia at the right base and an increase in the signs at the left base inside the region of her empyema.

For three weeks these symptoms persisted, until the thirty-seventh day when her temperature reached normal. During this time, she was studied carefully by a number of observers. The shadow to the right was seen in the fluoroscope

Fig. 7.—Mediastinal tumor removed at necropsy.

to be situated close to the anterior chest wall, and to pulsate, both of which features made a dilated aorta all the more probable, in spite of the negative Wassermann reaction and complete absence of syphilitic history. On the left side, a hilum shadow, rounded, rather sharply defined, appeared on rotation to be definitely behind the heart, which fact, in addition to the fact that the posterior mediastinum was clouded and her sepsis pronounced, made an abscess of the mediastinum seem likely, and aspiration was advised. It was decided, how-

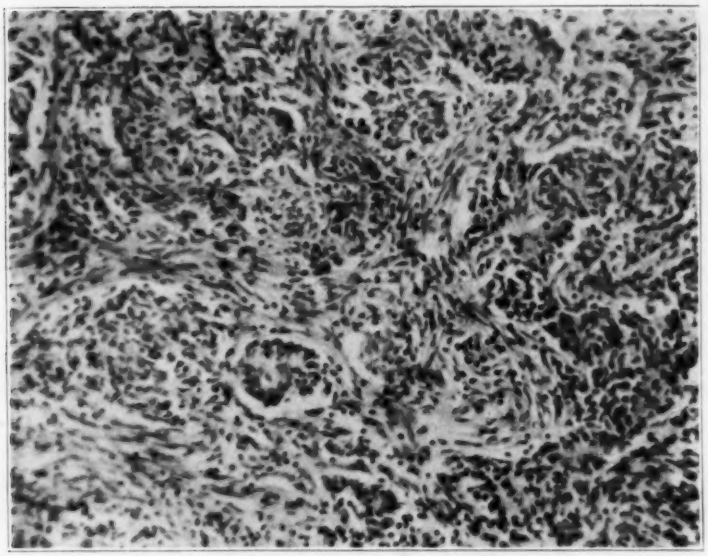

Fig. 8.—Mediastinal tumor as seen under lower power than Figure 9.

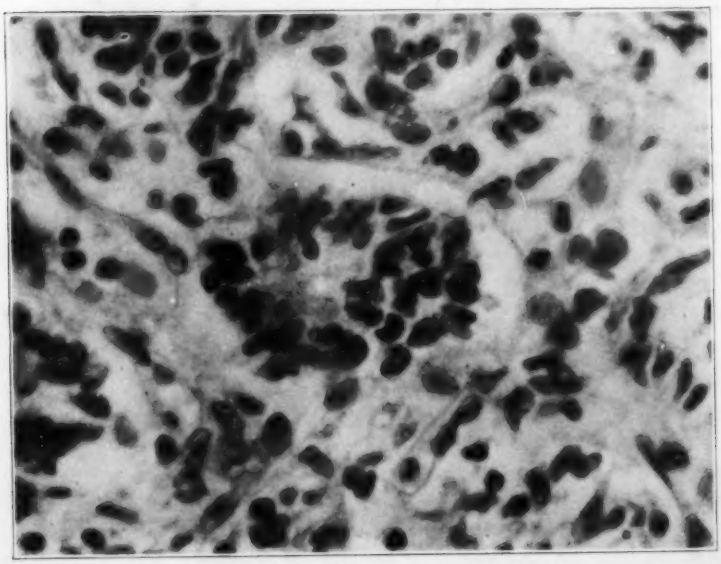

Fig. 9.—Mediastinal neuroblastoma showing a "rosette" form, as seen under high power.

ever, to postpone this, and the decision was justified by the temperature coming to normal three days later and the shadow on the left side clearing. The best explanation offered for what went on during these three weeks was that bronchopneumonia consolidations occurred in the right lower lobe, and in the central portion of the left lower lobe with considerable mediastinal cellulitis and lymphadenitis.

Subsequent plates showed disappearance of the pneumonic shadows, but persistence of the convex shadows as seen on admission.

Gradually, the patient regained enough strength to walk, in spite of a severe secondary anemia, and finally insisted on going home ninety-one days after the operation. Eleven days later, she was brought back to the hospital moribund, with a red cell count of less than a million and hemoglobin 15 per cent., and died in a few hours.

The necropsy findings, as recorded by Drs. Curtis, Von Glahn and Pappenheimer, bring out remaining points of especial interest.

The heart muscle was pale and showed fat infiltration. There were 400 c.c. of clear fluid in the right pleural cavity. On the left side was the bronchial fistula, much contracted, into which the descending main bronchus was found to extend directly.

The aorta showed no dilatation, no specific aortitis, only one small, intimal, fatty change in the transverse arch.

What was taken for a dilated aorta in the roentgenograms was a tumor, 10.5 by 8.5 by 3.5 cm., wider above than below, encapsulated, fibroid, not adherent to surrounding tissues, immediately behind the chest wall, in front of the arch and ascending aorta, with its right border closely resembling the curve that the ascending aorta takes in its ascent from the heart.

Its microscopic picture showed innumerable whorls and strands of cells that stained yellow with van Giesen's stain between trabeculae of fuchsin staining connective tissue collagen fibers. Here and there was an arrangement in "rosette" formation, with central network of fine fibrils. Dr. Pappenheimer made the following note: "I agree with Dr. Von Glahn and Dr. Curtis in the diagnosis of neuroblastoma; the arrangement of the cells and the character of the fibrillar stroma are very typical. This is a case of particular interest because of the unusual situation of the growth."

In 1910, Dr. Wright described twelve cases.[1] In 1914, Wahl[2] gave an exhaustive report on a case combining in one growth what he considers the three elemental types, ganglioneuroma, neuroblastoma and paraganglioma (or chromaffin tumor), as well as a most complete bibliography. None of the cases reviewed in the literature by Wahl were in the anterior mediastinum.

DISCUSSION ON PAPERS BY DRS. GRAHAM AND AUCHINCLOSS

Dr. J. J. Singer, St. Louis: I should like to take up the medical side of the problem. One can't help but feel that all these surgical cases have a medical side. Some one coming through our hospital recently asked how it was we had so many cases for cauterization, and we told him we looked for them, and the "looking" usually is in the hands of the medical men.

1. Wright, J. H.: J. Exper. Med. **12**:556, 1910.
2. Wahl: J. M. Res. **30**:205, 1914.

The great cry that we heard from the men who spoke on the cancer of the esophagus was to get the patients early. Well, to get the patients early in abscess of the lung you have to look to the medical man, and it is the medical man that needs more or less education.

We have tried, in chest work, at the Barnes Hospital and the Washington University to find these cases early. Unfortunately, a great many are not early when the patient presents himself, but we have found some shortly after the beginning of the trouble.

The first patient that Dr. Graham showed and the first patient that was operated on was a patient who came to me six weeks after the tonsillectomy, before there was any evidence of an abscess other than a little nodule in the hilum, but the history of tonsillectomy, fever and cough was of sufficient importance to us to wait and to expect an abscess, and abscess finally resulted.

Unfortunately, in this case, the cauterization wasn't done for a long time because no one had thought of a cauterization; Dr. Graham was out of the country at the time that the patient was sick, and she was operated on for an empyema that had developed following a pneumothorax, and the ordinary operation for abscess of the lung was instituted by a man who took Dr. Graham's place while he was away. After drainage of the abscess by a tube, the patient was just as miserable as she was before, and we were more or less without knowing just what to do but wait developments. The patient was so very miserable, in spite of the operation, that Dr. Graham conceived the idea of cauterization. The results have been, I believe, miraculous.

A lone abscess in a lung is a rare thing. It is very seldom that it is possible to have one single abscess in a lung. It is possible that, even in opening an abscess, when there are multiple abscesses, the small bronchi that open into this abscess may drain a rather large number of smaller abscesses; but usually the many abscesses are blind, and so, in analyzing the value of this operation, I feel that its main purpose is the removing of a large section of infected tissue and leaving open innumerable bronchi and bronchioles which are the drainage tubes, one might say, of the many tiny, even microscopic abscesses.

We found that many of these small abscesses are seen only by cauterization, and that a great many of these are blind, as far as drainage is concerned.

On the question of emboli, I don't know of any method that can be used to prevent embolism in cauterization. It has been brought out by Schloepfer that, if one inserts a needle into the chest wall, one is apt to put the needle into some blood vessels of adhesions and, thereby, let air into the general circulation; but, in cauterization, the tissue is all exposed, and air can be aspirated into many vessels.

Dr. Franz Torek, New York: Reference was made to the question of diagnosis of mediastinal tumor versus abscess. In the former, the very sharply defined outline of the mediastinal tumor would stand out more definitely than that of an abscess; in the case of an abscess, there would be gradual shading off of the shadow.

In reference to the result of Dr. Graham's method of treating lung abscess, it certainly is remarkable to get a result as nearly perfect, from a method in which he operates without the advantage of seeing, as one would with a wide open operation, and I can imagine that with each successive cauterization the difficulty must increase, because the size of the opening naturally must diminish in time. It also is very interesting that he has had so little trouble with hemorrhage.

DR. WILLY MEYER, New York: Dr. Graham's operation represents, to my mind, a tremendous step forward.

In some cases the lobe is completely adherent to the chest wall. We have seen patients at our hospital with a large opening leading into the clean abscess cavity of the lung. The question came up, in order to cure such a patient, should we loosen the lobe and do merely an additional thoracoplasty or perform lobectomy? It seems to me that in these cases the cautery pneumectomy is clearly indicated.

Dr. Auchincloss' case shows how necessary it is to remember the possible presence of a tumor in these cases of chronic suppuration.

DR. WILLIAM H. STEWART, New York: I should like to ask Dr. Auchincloss if roentgen-ray investigations were made in the lateral position in the differentiation between a dilated aorta and a tumor. We have found that the lateral position, erect and prone, will give us much more information than other positions. Stereoroentgenograms must be taken in all positions.

DR. EDWARD W. ARCHIBALD, Montreal: We are all familiar with the march of events in the treatment of chronic lung suppuration, particularly of bronchiectasis and we all know how we have been endeavoring to reduce the mortality. Lilienthal's series of thirty-two cases, published two years ago, gave a mortality of 50 per cent. or a little over, and that seemed prohibitive.

A year ago, I proposed an extensive thoracoplasty as a necessary preliminary to bringing out the affected lobe of the lung to the exterior in order to prevent empyema, pneumothorax and, if possible, mediastinitis. That procedure, which, I think, may still have its indications, might have lowered the mortality very considerably. But I wish now to align myself publicly, as I have already said privately to Dr. Graham, under his standard. It does seem to me that the previous work of Lilienthal and others in the treatment of chronic lung suppuration is to a very large extent superseded by this particular method, which not only offers a larger measure of safety but also apparently yields most excellent results.

DR. CARL A. HEDBLOM: I should like to add my word of appreciation of both papers. From the standpoint of pathology, Dr. Auchincloss' paper was certainly of great interest. I am particularly interested in the subject of cautery pneumectomy in suppuration. If it proves to be a safe method from the standpoint particularly of hemorrhages, I think it will represent the greatest advance that we have made so far in the treatment of generalized chronic suppuration of the lung.

I still feel that, for those cases of bronchiectasis in which the roentgenogram shows very little shadow, the extrapleural thoracoplasty is the operation of choice because it is a safe and effective operation; so far we have not had a single death in cases in which we have operated, and we have had a high percentage of symptomatic cures. I believe that, from the standpoint of the amount of risk, the results obtained and the amount of deformity, there is nothing better to offer the patient; but in cases in which there is associated multiple abscess with the bronchiectasis or in which there is a diffuse shadow, so that one is unable to determine the exact pathologic conditions, this method certainly is to be considered. I have tried it in four cases; I had hemorrhage in three. In one case, the hemorrhage was controlled with the pack; two patients died very suddenly; the bleeding was in the bronchial tree. In one case, the accident happened the seventh day, when my house surgeon and the nurse were doing a dressing. They were changing the pack; there was sudden, profuse hemorrhage through the wound and by mouth and, in the course of two or three minutes the patient was dead.

The other case was that of a man who had had a prolonged suppuration and was in poor condition, and there was a question as to how large a part respiratory failure played; but hemorrhage in the bronchi in that case was the immediate cause of death.

Now, these are only two cases out of four, against Dr. Graham's series, and I want to emphasize the point that I do not consider that such a small series of cases is any criterion, but I have raised the question with Dr. Graham many times as to what we can expect in the way of hemorrhages; if we can save 80 per cent. of the patients I think it is the operation of choice, because so far we have not been able to approach that percentage of cures by any other method.

It is a most suggestive and interesting contribution and one which I think merits careful consideration in the treatment of these cases.

Dr. Evarts A. Graham, St. Louis: Dr. Hedblom has raised the question of hemorrhage in cases of pneumectomy with the cautery. There is, of course, frequently a good deal of hemorrhage during the operation because large vessels are cut through with the cautery. I cannot understand, however, how there could be any difficulty about controlling this hemorrhage, because the blood pressure in the pulmonary artery is only from 15 to 20 mm. of mercury. This permits easy control by means of pressure. In fact, in not a single instance have I ever experienced any difficulty in controlling the hemorrhage which was produced by the cautery. It has been my custom to stop whenever I encounter serious hemorrhage, to pack, and to postpone any further operative work until about a week later. Usually the pack can be removed safely on about the third day, but, if any hemorrhage starts on removing the pack, it is a simple matter merely to replace the pack. As far as postoperative hemorrhage is concerned, I have never seen a single instance of postoperative hemorrhage from the lung after the use of the cautery. I did have one hemorrhage from an intercostal artery which, however, was easily recognized and controlled by clamping the spurting artery.

CHRONIC PNEUMONIA OR TUMOR OF THE LUNG

AN INTERESTING OBSERVATION OF INFILTRATION OF THE RIGHT UPPER LOBE

WILLY MEYER, M.D.
NEW YORK

It is recognized today that in refining the diagnosis in nonspecific affections of the lung with productive sputum, roentgenography and bronchoscopy in addition to the careful and complete clinical and laboratory examination of the patient, are of greatest assistance to physician and surgeon.

After the roentgen-ray examinations have shown haziness, fuzziness, infiltration, cavity formation with and without a fluid level on the stereoroentgenograms—in the erect and horizontal posture as well as in the lateral view—the trained bronchoscopist will give us further details. Having thoroughly evacuated the mucopurulent secretion from the bronchial tree by means of suction, he is in a postion to watch the appearance of the discharge at the very outlet of its source, at the respective bronchial opening, and also to detect, at the latter spot, granulations, ulcerations and stenosis of orifice or lumen; this visual observation then can be corroborated by intrabronchial instrumental manipulations.

Most important clinical data will be collected in this way, which, in conjunction with the results of the original examination, affords a degree of precision unheard of one or two decades ago.

Of course, physician and surgeon must have learned to segregate from the start. The carefully taken history and the general—call it rough—laboratory findings, must have led them on to the right track in their diagnostic endeavors.

In the interest of an eventually required surgical intervention, the surgeon's hope may be repeated at this juncture,[1] that competent roentgenologists will lend their efforts to a still more detailed localization of the diseased focus as to depth, direction and distance from a fixed spot or spots of the skeleton, within the rather vast area of the two sides of the chest cavity. True, laudable beginnings have been made; but reliable information at the hands of the roentgen-ray specialist, as compared to his able guidance in cases, for instance, of a foreign body in other regions than the chest, is still outstanding.

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1. Meyer, Willy: Observations on Lung Suppuration and Its Treatment, Arch. Surg. **6**:361-425 (Jan.) 1923.

Of course, it may be said that such endeavors are superfluous nowadays, in that the surgeon can with impunity open the pleural cavity, inspect and palpate the lung in order to determine the presence of infiltration of the lung tissue and of localized adhesions between pulmonary and costal pleura, and let his decision as to the required operative procedure rest on such data. It may further rightly be said that the surgeon can resect ribs in sufficient number and length in order to be guided in his search for the diseased area of the lung by the pink—yellow—and free mobility of the healthy lung as it shines through the costal pleura under bright illumination, in contrast to the bluish-black appearance of the depth of the operative field and immobility of the lung with the acts of respiration, when the surface of the true area of the inflamed lung tissue has been exposed. However, nobody can and will deny that work of this character and extent may complicate the surgical procedure and in many cases may reduce the chances for the patient's recovery. Whereas, if we knew greater details *with precision* through the work of the roentgenologist, the chances for recovery in the given case might accordingly be enhanced, particularly in weak and reduced patients. I am sure that every general surgeon who has added the inside of the chest to his daily work as well as the specializing thoracic surgeon, will have wished for this "intrathoracic advance" in roentgenography, an advance so direly needed. Should it come forth, it will mean another great step forward in the conservative treatment of suppurative diseases of the lung.

The foregoing refers to affections of the lung that show productive sputum. But what is the situation, if the sputum is a negligible factor in the clinical picture, if it amounts to less than 1 ounce (30 c.c.) in twenty-four hours, or is entirely missing, if repeated analysis of the sputum does not yield characteristic signs, if, in a reduced and weak patient, the differential diagnosis at last rests between tumor and chronic inflammation of a portion of the lung? A quick way of severing the Gordian knot, of course, would be to do an exploratory thoracotomy. But supposing there is unalterable opposition on the part of the patient, as well as his family, to all operative intervention, and supposing bronchoscopy does not appear to be indicated and, at the same time, represents an unnecessary strain for a debilitated patient above 50 or 60?

Here, continued and careful clinical examination and observation—best at the hospital—giving particular regard to the information furnished by regular thermometry; patience and perseverance in the laid down line of treatment, and the thorough analysis and study of the result of the therapeutic measures, as they appear from week to week, will be the torchlight of hope in the given case. Proper expert interpretation of the roentgenograms, taken at regular intervals, will assist in determining whether the course of the medical man is right or wrong.

A case of the latter type, recently observed, which elicited a great deal of discussion, pro and con, with respect to an eventual surgical intervention has furnished the incentive for the presentation of this paper.

REPORT OF CASE

S. R., a man, aged 53, first seen by me in consultation at his home, Oct. 27, 1923, had always been well up to September, when he felt weak and complained of a "whistle in his chest, when he breathed." Two weeks prior to my visit he had developed all the signs of a bronchial pneumonia of the right upper lobe.

Fig. 1.—Roentgenogram, taken at end of October, suggesting the diagnosis of an interlobar empyema on the right side, particularly in the light of the clinical history and phenomena.

With the usual treatment at the hands of his family physician, a drop of the fever to almost normal was noticed on the seventh or eighth day after the onset of the severer pathologic symptoms. Shortly thereafter, the temperature rose again.

A nurse was engaged, and a regular chart started. The fever was hectic in character, touching the normal level in the morning and rarely rising above 103 F. in the afternoon. A bedside roentgenogram showed a non-infiltrated right upper lobe and a number of opaque areas, running in a rather horizontal, outwardly slightly oblique upward line, corresponding to the space between the right upper and middle lobe (Fig. 1). The roentgenologist,

in consultation with the attending physician, interpreted the picture as right interlobar empyema, and suggested, should aspiration be deemed indicated, introducing the needle in the fifth interspace posterior to the axillary line. This was done on the morning of the day I saw the patient, but no pus was found. The result of percussion and auscultation at my hands was rather negative; there was slight dulness anteriorly and posteriorly, corresponding to the right upper lobe, but hardly a râle; there was no sputum that could be seen and investigated. The sputum, very scant, had shown a few streaks of blood on previous days and some mucopus without tubercle bacilli. I concurred with the probable correctness of the diagnosis of a beginning interlobar empyema, particularly on the basis of the roentgenogram and the curve of

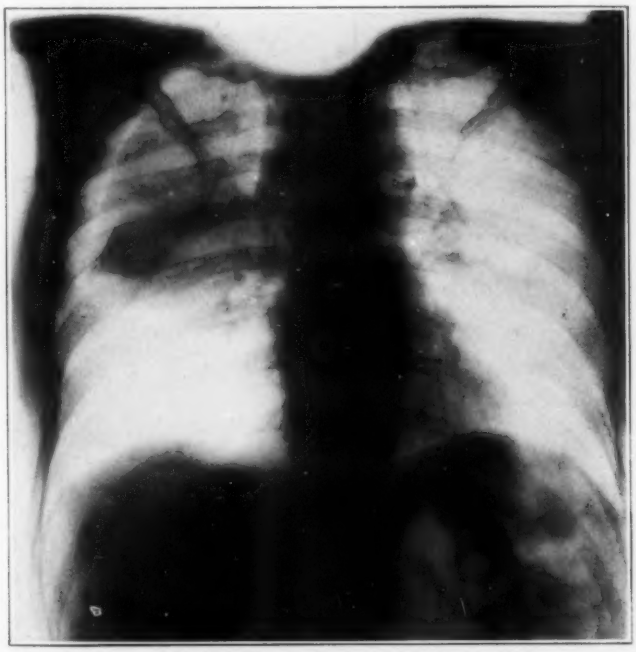

Fig. 2.—November 5, an unintended slight artificial pneumothorax following exploratory puncture, done with negative result.

the fever, and advised a stereoscopic roentgen-ray examination at the roentgenographer's office with continued careful clinical observation at home. The stereoroentgenogram was taken five days later, November 5. It showed a pneumothorax in the upper portion of the chest, producing partial collapse of the superior lobe, which, on the film, appeared opaque and infiltrated (Fig. 2).

A change in the patient's general condition soon caused worry among the members of the family, and he was admitted to the private division of the Lenox Hill Hospital under my care, November 6.

The pulse was slow, less than 90 a minute; the temperature was of the same hectic type as before, but about 1 degree lower than previously recorded, the reduction being probably due to the unintended artificial collapse of the affected portion of the lung; respiration was not above 25; cough was rare.

The sputum was very scant, containing some mucopus, and was now and then streaked with blood; it was negative for tubercle bacilli and tumor cells. The Wassermann reaction was negative. There was no tumor in the rectum. The urine analysis revealed no albumin and no sugar. The blood count showed a low percentage of polymorphonuclear leukocytes; the white blood cells totaled 15,000. Inspection, palpation, percussion and auscultation pointed to an inflammatory affection of the right upper lobe. The alternatives in differential diagnosis were: interlobar empyema; nontuberculous, subacute pneumonia, and tumor formation. Examination of the blood sugar equilibrium pointed to the absence of malignancy.[2]

Fig. 3.—Pneumothorax and infiltration of right superior lobe increased; exposure in supine posture of patient, November 13.

The treatment was expectant; rest in bed; care that there should be the proper amount of intake and sufficient output; a Priessnitz dressing over the upper portion of the right side of the chest, day and night; a follow-up of the intrathoracic condition clinically and roentgenographically, observing the gradual disappearance of the accidental pneumothorax. The necessity of patience and perseverance was persistently impressed on the patient's mind.

Dr. William H. Stewart reported that the roentgenograms taken, October 30, showed distinctly a pneumonic process in the upper right chest. Observations

2. Observations at Lenox Hill Hospital showed about 80 per cent. of reliable results from this type of examination, at least in intra-abdominal cases.

Fig. 4.—The same condition as in Figure 3, with the patient in lateral posture, November 13.

Fig. 5.—November 16, the same condition as in Figures 3 and 4 with the patient in erect posture, anteroposterior view; pneumothorax increased; adhesions at the apex particularly well marked.

made, November 5, before admission as well as those made at the Lenox Hill Hospital, revealed what was apparently a solid right upper lobe; there was some air in the upper portion of the right pleural cavity with some recedence of the lung from the chest wall (Figs. 3 and 4).

November 16, according to all the roentgenograms taken, there was collapse of the upper right lobe, due to pneumothorax; bands, pleuritic adhesions were preventing complete collapse at the apex (Fig. 5); the shadow of the collapsed lung was very smooth and dense, which would seem to indicate either merely a collapsed, air-free lung or some remaining inflammatory process. There was nothing in the roentgenogram that would suggest an interlobar empyema. The lower lobes of the right lung had not collapsed, which was

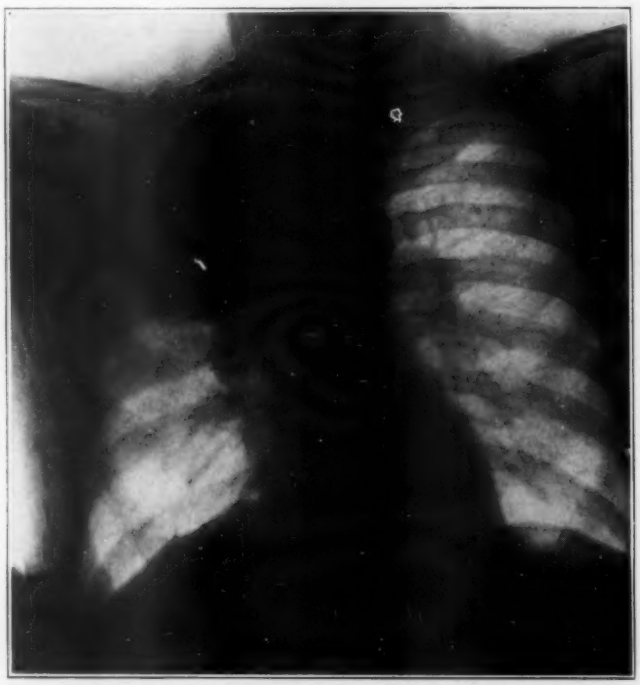

Fig. 6.—Pneumothorax decreased; roentgenographic diagnosis: tumor or sacculated fluid rather than chronic pneumonia.

evidently due to a blocking of the lower portion of the right pleural cavity with adhesions. The shadow seen in the film taken on November 5, posteriorly, suggested a possible interlobar empyema; the one taken anteriorly, however, and a subsequent series made seemed to exclude this lesion. The shadow, as seen in the anterior film of October 30, seemed to indicate that the process limited itself to the lower portion in the upper right lobe.

November 20, fluoroscopic examination, as well as the stereoroentgenograms in both anterior and posterior positions, revealed that the upper right pneumothorax had considerably increased. The entire right upper lobe was collapsed and, fluoroscopically, it acted much like a carnified lung; a pus sac could not be recognized.

December 4, the collapsed upper lobe of the right lung was considerably expanded, and the pneumothorax had greatly diminished. The lung substance was much more dense than noted on the previous examination; the outline seemed to be rather irregular (Fig. 6). The roentgenographic findings favored a sacculated fluid or lung tumor rather than chronic pneumonia. The indications seemed to be for further needling in an attempt to obtain fluid. A side view taken failed to show adhesions of the pleural leaves in front; aspiration was, therefore, not carried out.

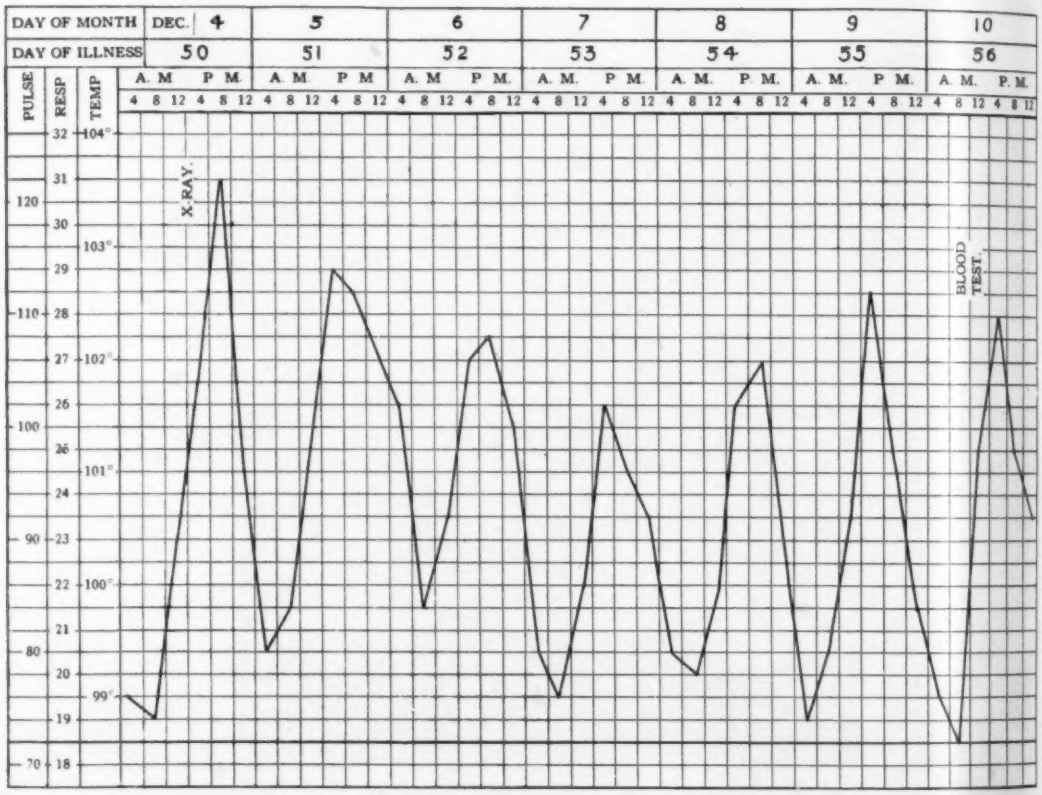

Fig. 7.—Chart of temperature of week, from December 4 to 10, showing the continued hectic type of fever. All temperatures are rectal. White blood cells totaled 16,200; polymorphonuclears were 86 per cent.; mononuclears, 14 per cent.

December 12, the right upper pneumothorax had almost completely disappeared; there was what was apparently a solid upper lobe, the lower border of which was sharply outlined.

December 7, there were signs of pleurisy over the upper part of the right lower lobe, posteriorly. Physical examination, December 9, revealed the slight lagging behind of the right side of the chest on deep inspiration. On palpation, over the right chest, anteriorly, just below the clavicle from the midline outward to about the midclavicular line, vocal fremitus was somewhat diminished.

Over the right base, it was slightly increased posteriorly with a very slight friction rub perceptible. On percussion, in the supraclavicular fossa and in front of the chest over the right upper lobe, just below the clavicle from the midline outward to the midclavicular line, there was a flat percussion note with an admixture of slight tympany; externally to this, in front, there also was flatness, while posteriorly, over the apex, there was resonance, as elsewhere over the lung.

On auscultation, over a very much circumscribed area in the supraclavicular fossa and below the clavicle from the midline outward and down to the level of the third rib in the midclavicular line, there was bronchial breathing, voice sound and whispered sound, but no râles on coughing. At the base of the right lung, posteriorly, from the angle of the scapula downward and as far forward as the posterior axillary line, a loud friction rub could be heard on inspiration and expiration, but no bronchial breathing. Everywhere else over the lung, there was normal vesicular breathing; voice sounds were easily audible. There were no physical signs of pneumothorax. The temperature was of the same hectic character as previously, dropping to normal with heavy perspiration early in the morning, and rising regularly to from 102 to 103 F. and more, in the afternoon (Fig. 7), often with a distinctly chilly sensation; there was hardly any cough or expectoration.

December 11, consultation was held with Dr. O. M. Schwerdtfeger of the medical division who inclined to the diagnosis of an inflammatory process of the right upper lobe rather than a neoplastic one and advised alternating daily treatment with Alpine sunlight and diathermia in conjunction with the same local and general tonic and stimulating regimen as carried out so far.

This advice was promptly acted on, and the treatment was carried out regularly and faithfully in the physiotherapeutic division of the Private Building of the Lenox Hill Hospital. After two treatments, queer to report, one of diathermia and one of Alpine sun, the afternoon temperature dropped 2 degrees (Fig. 8); however, there were occasional rises above 101 or 102 F.; the slight cough with expectoration slowly increased; the latter contained bright blood for the first time subsequent to the eighth physiotherapeutic application, with the highest temperature of the day at 100.4.

In the course of the last three weeks, the white blood corpuscles had been counted usually around 16,000; polymorphonuclears increased from 72 to 86 per cent. (December 10).

December 11, white blood cells numbered 22,000; polymorphonuclears, 86 per cent.; December 15, white blood cells, 20,400; polymorphonuclears, 78 per cent.; December 22, white blood cells, 16,900; polymorphonuclears, 76 per cent.

At a second examination of Dr. Schwerdtfeger's December 23, he noted that, in the right lung, anteriorly down to the third rib was dulness; near the sternum at the second interspace was an area of tympany. Posteriorly, there was dulness over the entire lung, quite marked at the apex and gradually lessening; at the base, the percussion note was about normal. In the infraclavicular region, the respiratory murmur was bronchovesicular; at the angle of the scapula was a large area of pleural friction rub; only a few râles were heard in the infraclavicular region and they were obtainable only on deep inspiration after coughing; the heart was not displaced; the left lung was normal; there were no evidences of pressure or enlarged glands. The physical signs were about the same as two weeks before, except that there was some bronchial character to the breathing in the infraclavicular region; the general condition seemed to be greatly improved.

The anatomic diagnosis was a chronic inflammatory process of the upper half of the right lobe with thickening of the pleura and roughening of the pleura over the entire lung posteriorly. The clinical diagnosis was chronic pneumonia or neoplasm of the right lung. The physical signs could fit either condition. There were no characteristic signs of neoplasm, such as pressure symptoms, glands, or cardiac displacement, nor were there any areas showing complete solidification of lung tissue. The general evidences of receding inflammation (fewer leukocytes), the pleurisy and the slight improvement in physical signs rather than a progression made the diagnosis of chronic or unresolved pneumonia the more probable. Continuation of the physical treatment was urged.

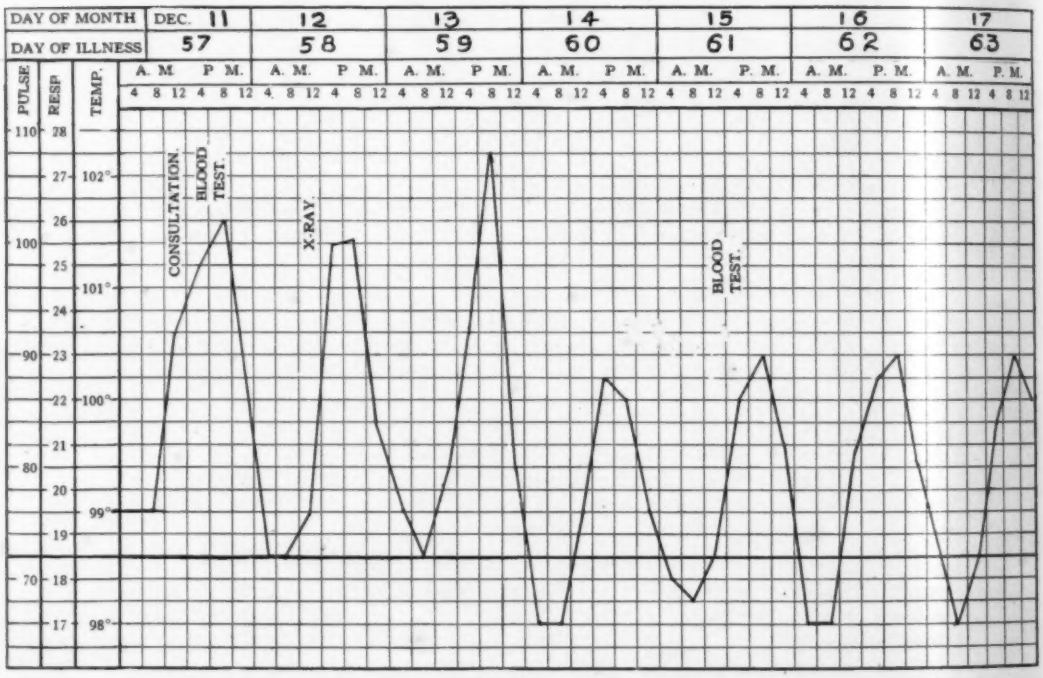

Fig. 8.—Chart of the week following that of Figure 7, December 11 to 17, showing the remarkably prompt and lasting drop in the temperature (December 14) after the start of physiotherapy. December 11, white blood cells totaled 22,000; mononuclears were 14 per cent.; polymorphonuclears, 86 per cent. December 12 to 15, white blood cells totaled 20,400; polymorphonuclears were 78 per cent.; mononuclears, 22 per cent., and systolic blood pressure 110, diastolic 90.

Beginning, December 27, the afternoon temperature never reached 101 F. and dropped under 100, Jan. 3, 1924, for the first time, continuing normal after that, with the exception of January 12-13, when the highest recorded was 101.4 and 100 F., respectively (Fig. 9).

The roentgen-ray examination report stated that the lower border of the consolidated upper right lobe seemed to show some evidence of aeration. The

distance from the apex to the lower border of the density seemed less than at the last examination. There was no evidence of a pneumothorax (Fig. 10). These findings would seem to indicate some improvement in the condition. Figure 10, one of the stereoroentgenograms, gives no adequate idea of the transparency of the lobe on stereoscopic inspection.

Beginning, January 9, four weeks after starting the roentgen-ray and hyperemic treatment of the lung, the expectoration contained bright red blood in varying quantities almost daily. January 13, white blood cells totaled 15,800; polymorphonuclears were 72 per cent.; the red blood cells numbered 3,480,000, and the hemoglobin was 60 per cent.

Dr. Schwerdtfeger noted, January 13, that the general condition was excellent. In the right lung, posteriorly, physical signs on both sides were normal and the same; anteriorly, there was dulness in the upper half of the lung; the dull

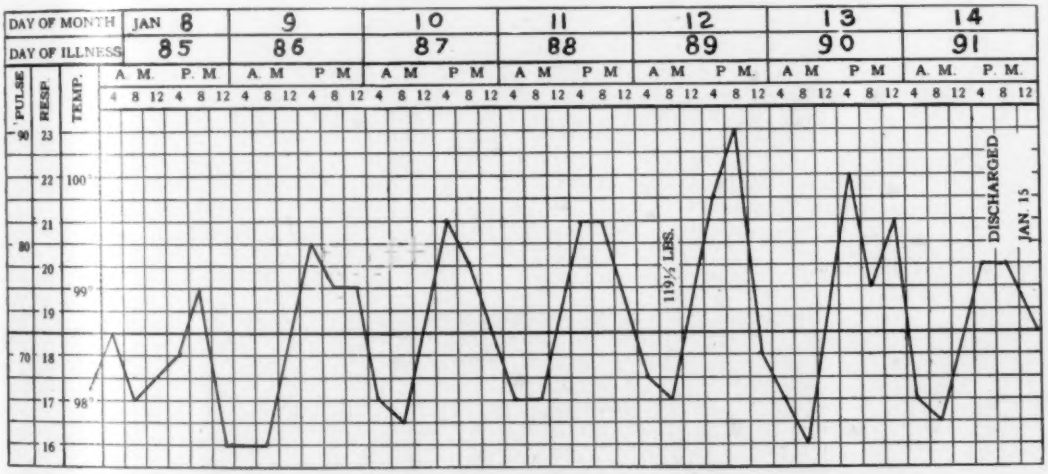

Fig. 9.—Temperature chart of week, January 8 to 14; temperature normal now, except on two days. January 13, white blood cells totaled 15,800; polymorphonuclears were 72 per cent.; red blood cells totaled 3,490,000; mononuclears were 28 per cent., and hemoglobin, 60 per cent.

tympany next to the sternum between the second and the third ribs was less marked; in the infraclavicular region, the bronchovesicular breathing was only very slightly present; at other places, anteriorly, the breathing on both sides was the same, i. e., normal; the heart was not displaced, and there were no pressure symptoms. The signs of inflammatory infiltration in the upper half of the right lung had greatly diminished since the examination three weeks before.

January 15, the patient was discharged at the request of his family, in good general condition and with an increase in weight. About one hour before he left the hospital, he expectorated almost 4 ounces (118 c.c.) of diffusely dark red, bloody sputum, the largest amount during his entire stay.

The advice to continue the physical treatment in an ambulatory way, at regular intervals, was not accepted; the patient wished to go to the country,

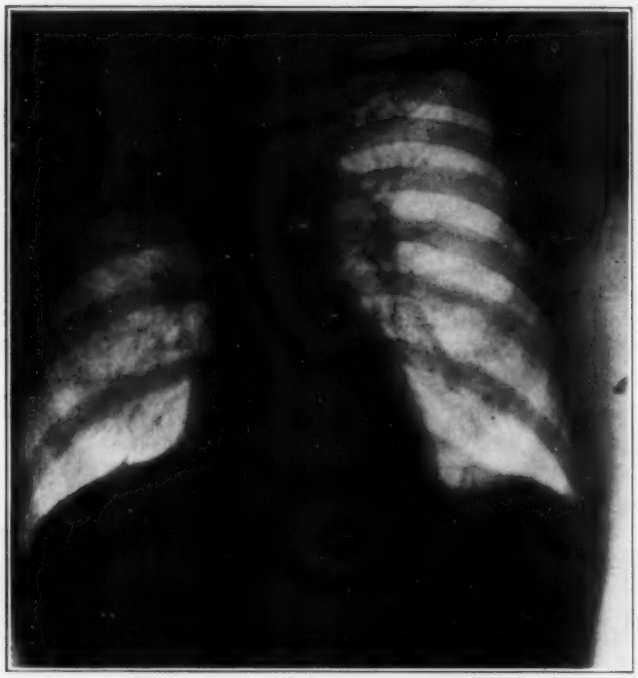

Fig. 10.—January 4, the entire right upper lobe more transparent in the stereoroentgenograms, suggesting the gradual disappearance of the chronic pneumonic infiltration under continued alternating use of diathermy and Alpine sun.

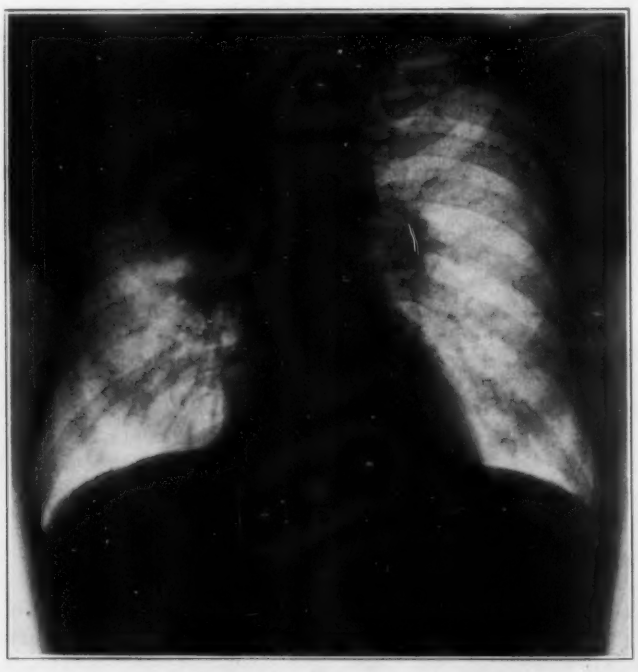

Fig. 11.—Appearance of the right upper lobe, March 5, after patient had gained almost 20 pounds (9 kg.); there can be no doubt that the malignant tumor got the upper hand.

where he stayed for two months, gaining over 15 pounds (6.8 kg.). The temperature rose to from 101 to 102 F. on rare occasions, and the scanty sputum showed blood now and then.

March 6, the roentgen-ray and fluoroscopic reexamination report stated that the process in the right upper lobe was much the same as when the patient was last examined at the hospital. The case was felt to be one of malignancy and it was hoped to prove or disprove this diagnosis by further observation (Fig. 11).

April 4, after clinical examination, Dr. Schwerdtfeger reported that the patient complained of pains over the lower ribs anteriorly and posteriorly, and of general weakness. There was the same dulness from the clavicle down to the third rib present as before; over it slightly exaggerated vesicular breathing. There were no glands felt, and no signs of pressure; hemoglobin was 90 per cent.; the weight was 132 pounds (59.9 kg.).

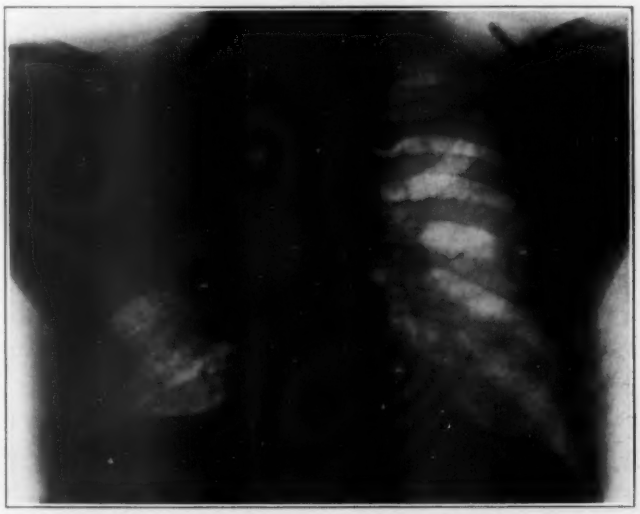

Fig. 12.—Marked increase in the growth of the pulmonary neoplasm, May 10.

April 23, there was pain in the chest and upper abdomen; the sputum often was bloody; there was distinct, harsh tubular breathing, anteriorly, in the second intercostal space, next to the sternum and in the interscapular space, likely due to the pressure on the trachea. The hemoglobin was 73 per cent.; the weight, 126 pounds (57.2 kg.).

May 8, the patient was again seen by me. He had lost 10 pounds (4.5 kg.), and complained of great pain in the region of the liver. The latter was not enlarged, and the pain was probably due to intercostal neuritis, dependent on the involvement of the intercostal nerves by the advancing tumor formation, which was clearly visible in the roentgenogram taken, May 10 (Fig. 12). The sputum was mucopurulent, and increased in quantity (probably beginning bronchiectasis complicating the neoplastic disease).

Today, in the light of further developments, a combination of a chronic inflammatory process of the lung *with* tumor formation is the most plausible diagnosis in this case. Physiotherapy with the Alpine sun and diathermy reduced

or removed the inflammatory infiltration; but, of course, the tumor continued to grow, although malignant cells never were found in the sputum. Beginning early in May, the effect of deep roentgen-ray treatment was tried.

During the first days of June, the patient developed high fever with difficulty in breathing. He died, June 12. No necropsy was done.

I regret that I did not insist on bronchoscopy shortly after the patient's admission to the hospital; it was the only modern examination which was not employed, and might have earlier cleared up the additional diagnosis of "tumor." Perhaps a small growth could have been seen within the bronchus; but in view of the very scant sputum and the greatly reduced general condition of the patient, it did not seem to me to be indicated at that time.

Later on, the slight hemoptysis on the day of the patient's discharge from the hospital was very suspicious, although the subsequent remarkable gain in weight was again reassuring.

SUMMARY

1. There is great difficulty in differentiating between chronic inflammation and tumor formation in the lung in certain cases.

2. There is the possibility of a combination of the two diseases in the early stages of a malignant tumor.

3. A typical, hectic type of fever in chronic affections of the lung in the absence of purulent sputum, so often seen in pulmonary tuberculosis, may occur also in chronic nontuberculous inflammation of the lung, as well as in tumor formation.

4. The therapeutic value of physiotherapy—Alpine sun and diathermy—in chronic inflammatory affections of the lung is great.

5. The possibility that the neoplastic infiltration of the right upper lobe also was benefited by this treatment, at least temporarily, in this case, cannot be denied.

6. It is advisable in subacute and chronic affections of the lung, in which a precise differential diagnosis is difficult, to insist on employing *all* the modern means at our disposal, i. e., bronchoscopy also, if it can be assumed that the patient's condition will likely not be aggravated by this examination.

PRIMARY TUMORS OF THE LUNG*

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Primary tumors of the lung are surprisingly frequent. All newer writers agree that pulmonary cancer is increasing. The increase is not only apparent but real.

The frequency of pulmonary tumors in newer clinical statistics might be explained by greater diagnostic accuracy; however, a similar increase is noted in necropsy statistics, and this cannot be accounted for otherwise than by matters of fact. The cause of the increase is no clearer than is the cause of cancer. Perhaps postinfluenzal changes have something to do with it.

In 1886, Fuchs calculated that primary cancer of the lung formed 0.06 per cent. of 12,307 Munich necropsies. Ewing (1922) states that, in deaths from cancer, the lung was found to be the seat of the primary tumor in about 1 per cent.; other statistics place the lung as being affected in from 1 to 2 per cent.; Berlin statistics give 3.4 per cent. (Karrenstein, 1908). Drs. Randolph and Rusk [1] gathered twenty-two cases of cancer of the lung from the University of California Medical School department of pathology since 1916, 1.36 per cent. of all necropsies

Of the twenty-seven cases discussed in this report [2] eight are based on necropsies done in the Stanford service at the San Francisco Hospital since 1920, i. e., 3.9 per cent. of a total number of 206 necropsies, and 20 per cent. of forty deaths due to cancer. This extraordinarily high proportion is explained by the fact that many patients with ulcerating tumors were transferred to the Relief Home; their necropsies do not appear in the records of the San Francisco Hospital.

But eighteen of the twenty-seven cases have fairly complete records; of the remaining nine there are more or less detailed necropsy protocols, but no clinical histories. Twelve of the eighteen patients under clinical observation were seen by me in person. I have to thank Dr. H. P. Hill for sending me two of them. For the report and the necropsy specimens of one, I am indebted to Dr. R. B. Scheier, to Dr. W. W. Boardman for another, and to Dr. A. S. Adler for a third. It is quite likely that

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* From the division of surgery, Leland Stanford Junior University School of Medicine.

1. Personal communication to the author.

2. Three, perhaps five, additional patients have been seen since this report was read.

Case	Age	Duration of Symptoms	Cough	Sputum	Blood	Pain	Dyspnea	Effusion Pleurisy	Loss of Weight, Pounds	Fever	Wassermann Reaction	Clinical Diagnosis
1	47	14 mo. dyspnea and cough; 2 mo. worse	+	0	0	+	+	Effusion	9	98.6 F.	+++	Bronchostenosis aneurysm; spontaneous pneumothorax and empyema; carcinoma of pleura
2	54	2 months	+	0	0	+	+	Dry adhesions	÷	103	+++	Bronchostenosis from aneurysm; interlobar empyema
3	59	Lost weight; 4 mo.; no symptoms	0	0	0 Later +	0	0	0	16	98.8	—	Pulmonary neoplasm (from roentgen ray)
4	46	5 mo., "cold"	+	Heavy white mucus	0 + with abscess	+	..	0	20	101	—	Pulmonary neoplasm; abscess
5	47	1 year pain; worse, 4 mo.; much worse, 2 mo.	± Slight, later +	± Slight, later +	0 Later +	+	0	Local adhesions	..	98.6	—	Abscess; tumor aneurysm; tumor (operation)
6	54	2 mo., cough; blood, 1 wk.	+ Slight	Little	+	0	0	0 Later +	0 Later +	98.6	— — —	Neoplasm
7	51	2½ mo., diarrhea, cough? tuberculosis +	+	?	0 later + died, hemrhage	0	?	Effusion; small, strong adhesions	18 (diarrhea)	99	?	Emphysema; chronic tuberculosis; pneumoconiosis
8	60	13 mo., fever; "flu," cough	+	+	+	+	0	0	20	+	?	Bronchiectasis; multiple abscess right lower lobe
9	53	10 mo., cough and sputum	+	+	+	0	0	Adhesions	30	100	—	Empyema right base; bronchiectatic abscess
10	69	9 months	+	0 Later +	+	+	+	Effusion	20-30	100 R.	—	Lung tumor
11	53	1½ months	+	+	0 Later ?	0	0	Strong adhesions	?	101.5	+++	Lung tumor; syphilis
12	..	2 days	+	Later	+	?	Attacks, later	Effusion	+	Normal	?	Aneurysm
13	59	Cough, 8 mo.; swelling over right clavicle, 3 months	+	+	+	0	0	Adhesions	20	102	— — —	Pulmonary tuberculosis; tumor clavicular region (Grawitz metastasis ?)
14	68	Pain, 2 mo.	+	+	+	+	0	Adhesions right; left pleura free	+	100	—	Thoracic aneurysm
15	78	Pain, 6 wk.	+	+	0	+	+	100 c.c. right; left pleura obliterated	?	101	—	Bronchopneumonia lower left; old tuberculosis bilateral
16	55?	1 year	0	0	0	+	+	2,000 c.c. fluid	60	+	—	Phlebitis; later tumor
17	67	General paralysis; no tumor history	+	+	+	0	0	Adhesions	?	105	Spinal fluid +++ blood —	General paralysis; consolidation lung, tuberculosis ?

Roentgen-Ray Examination	Operation	Lived in Hospital	Necropsy	Microscopic Examination	Metastases, Clinical	Metastases, Necropsy	Roentgen-Ray Diagnosis	Grounds for Diagnosis
Dense shadow left chest	Gland removed	5 wk.	Carcinoma whole left lung	Squamous cell	Supraclavicular	0	Consolidation, left; incorrect	Biopsy gland; correct
Upper pleura thickened; thickened hilum; aneurysm descending aorta	0	4½ mo.	Carcinoma upper left bronchus; metastasis; bronchiectasis upper left with abscesses	Squamous cell	0	Prostate; spleen; liver; retroperitoneal glands; peribronchial	Aneurysm; incorrect	Roentgen-ray examination; dulness left; incorrect
Pulmonary neoplasm; large 10 cm. round tumor central part left lung	0	11 mo.	0	No specimen	..	No necropsy	Neoplasm; correct	Roentgen-ray examination; hemorrhage; correct
Neoplasm; abscess	Radium + abscess	2½ mo.	Carcinoma bronchus right hilum	Squamous cell	0	Peribronchial glands; heart	Neoplasm; correct	Roentgen-ray examination; operation; correct
Abscess; tumor aneurysm	Thoracotomy	7 mo.	Carcinoma bronchus left lower lobe	Squamous cell	0	0	Abscess; tumor; aneurysm; correct	Roentgen-ray examination; operation; correct
Neoplasm	0	14 mo.	Carcinoma left upper lobe	Medullary carcinoma	0; later, liver	Liver; liver hilum glands; lung hilum; multiple lung	Neoplasm; correct	Hemorrhage; roentgen-ray examination; correct
Heavy shadow and peribronchial infiltrate left	0	11 mo.	Carcinoma left main bronchus	Squamous cell	0	0	Heavy shadow left upper; peribronchial infiltrate; incorrect	Consolidation left base; tuberculous sputum; incorrect
Shadow	Lobectomy	1 day	Sarcoma right hilum	Round cell sarcoma	0	Pulmonary	Bronchiectasis; incorrect	Roentgen-ray examination; consolidation right base; incorrect
Pneumothorax right ?	Drainage abscess	2½ mo.	Carcinoma right lower bronchus; bronchiectasis	Squamous cell	0	Heart; mediastinum	Pneumothorax; incorrect	Consolidation right base; incorrect
Infiltrate left lung ext. from hilum	Biopsy	10 days	0	Gland (supraclavicular) medullary carcinoma	Supraclavicular; rib	0	Infiltrate left lung extending from hilum	Glands; roentgen-ray examination; rib metastases; correct
Consolidation lower two-thirds right upper lobe	Gland removed	22 days	Sarcoma right upper bronchus	Lymphosarcoma	Supraclavicular	Cerv. mediast. retroperit.	Consolidation right upper; possibly interlob. empyema	Supraclavicular glands; roentgen-ray examination; correct
Dilatation thoracic aorta; aneurysm	0	6 mo. observation	Carcinoma right bronchus, hilum	Squamous cell carcinoma	Mediastinum	Heart; visceral pericardium; mediastinal glands	Aneurysm; incorrect	Roentgen-ray examination; incorrect
Heavy infiltrate mediastinal gland	Gland removed	13½ mo.	Carcinoma left apex; tuberculosis left and right apexes	Squamous cell	Supraclavicular glands; tumor left hypochondrium	Supraclavicular, peribronchial, mediastinal, retroperitoneal and mesenteric glands; heart	Mediastinal glands; incorrect	Metastasis; supraclavicular gland; incorrect
Sacculated aortic aneurysm external to right lung field	—	6 wk.	Carcinoma upper and middle right, right main bronchus	Squamous cell	Cervical glands	Stomach; pancreas; peribronchial glands; kidney; chest; liver; aorta	Thoracic aneurysm; incorrect	Roentgen-ray examination; enlarged heart; incorrect
Pneumonia, lower left	—	24 days	Carcinoma lower left bronchus	Medullary carcinoma; adenocarcinoma	No note of glands	Heart; pancreas; liver; kidney; suprarenals; right lung; peribronchial, mediastinal glands	Pneumonia; old tuberculosis; incorrect	Roentgen-ray examination; clinical findings; incorrect
Dense shadow, lower right	—	....	Carcinoma lower right bronchus	Adenocarcinoma	Pleura	Peribronchial gland; pleura; liver	Pleural exudate; partly correct	Tumor shreds in pleural exudate; correct
0	0	11 days	Carcinoma left hilum bronchus	Squamous cell	0	0	0	Clinical findings; incorrect

Analysis of Twenty-Seven Cases

Case	Age	Duration of Symptoms	Cough	Sputum	Blood	Pain	Dyspnea	Effusion Pleurisy	Loss of Weight, Pounds	Fever	Wassermann Reaction	Clinical Diagnosis
18	55	2 months	?	?	?	?	+	3,300 c.c. left; dense adhesions right	+	?	?	Malignant tumor, lung
19	36	1 month?	?	?	?	+	?	Empyema	+	+	—	Carcinoma; metastases liver, skull, sternum, lungs
20	48	3 years?	+	+	+	?	+	Adhesions	+	?	+	Pulmonary syphilis
21	70	?	?	?	?	?	?	?	?	?	?	?
22	?	?	?	?	?	?	?	?	?	?	?	?
23	?	?	?	?	?	?	?	?	?	?	?	Aneurysm ?
24	Museum specimen; no history		..	..	..	..	..	..	..	..	..	.....
25	Museum specimen; no history		..	..	..	..	..	..	..	..	..	.....
26	Museum specimen; no history		..	..	..	..	..	..	..	..	..	.....
27	51	Cough, 7 mo.	+	+	+	+	—	1,000 c.c.	20	100	—	Pleurisy with effusion; neoplasm

even more cases were observed than this series includes: that some were discharged undiagnosed and that others died without having mistaken diagnoses of pulmonary tuberculosis, lung abscess or bronchiectasis corrected by necropsy. A few observations made prior to 1915-1916 have been lost; all records of the San Francisco Hospital up to this date having been destroyed.

The series includes only primary tumors of the lung itself; I have not included tumors of the mediastinal glands, metastatic cancers and cancers of the pleura. The diagnosis has been verified in every case but two by sections taken from the tumor tissue, and in fifteen of the eighteen by necropsy. It is a pleasure to acknowledge the services of an energetic house staff in securing so many necropsies: nine out of ten is a good record for a city hospital.

Cancer of the lung occurs much more frequently in men than in women: Adler gives the ratio as 332: 121, or about 3: 1. The sex is known in twenty-five of our cases; of these, twenty-three were males,

of Lung Tumor—(Continued)

Roentgen-Ray Examination	Operation	Lived in Hospital	Necropsy	Microscopic Examination	Metastases, Clinical	Metastases, Necropsy	Roentgen-Ray Diagnosis	Grounds for Diagnosis
?	—	12 days	Carcinoma lower left lobe (bronchus)	Carcinoma, medullary ?	?	Peribronchial glands	?	Bloody pleural exudate; correct
0	Skin nodule chest excised	10 days	Carcinoma hilum left (bronchus)	Medullary carcinoma; endothelioma?	Skull, sternum, liver	Cervical, peribronchial, mediastinal glands; kidney; ovary; pancreas; thyroid; skull; chest wall; pericardium	?	Multiple metastases; partly correct
0	—	?	Carcinoma hilum left (bronchus)	Carcinoma, medullary ?	0	Upper anterior mediastinum; heart; pericardium; kidney	—	Clinical symptoms; syphilis; incorrect
0	?	?	Carcinoma upper left bronchus	Squamous cell	?	Peribronchial gland	0	0
0	0	?	Carcinoma left hilum; carcinoma bronchus	Stratified cylinder	?	Peribronchial and mediastinal glands; pleura; heart; kidney	0	0
Aneurysm?	—	?	Carcinoma bronchus (hilus) right ? left ?	Epithelial cell	Diabetes	Liver; pancreas	Aneurysm?	Roentgen-ray examination; incorrect
.....	..	..	Squamous cell	.....	.....	.....	.....	.....
.....	..	..	Medullary carcinoma	.....	.....	.....	.....	.....
.....	..	..	Medullary carcinoma; endothelioma?	.....	.....	.....	.....	.....
Heavy density lower left; two oval mediastinal shadows	—	13 + 19 days	0	0	Cervical glands?	0	Neoplasm	Roentgen-ray examination; bloody exudate

i. e., there were about twelve males to one female. Their ages lay between 36 and 78, and were distributed as follows: 30-40, one; 40-50, four; 50-60, ten; 60-70, four, and 70-80, two.

The histories are of much interest. They throw a sad light on our diagnostic and therapeutic abilities, and sadder still, they seem to show no way out of our perplexities. In reviewing them, we can see how we might occasionally have made better diagnoses, but only one, perhaps two of the eighteen seem to have had the slightest chance for a cure at any time during the course of their observation.

The symptoms might be divided into two groups: those arising from tumors of the hilum or the main bronchi, and those arising from more peripheral tumors. Those patients whose tumors were at or near the hilum suffered from symptoms of tracheal irritation, mainly a distressing, barking, paroxysmal cough, with little sputum. Those whose tumors occluded a large bronchus showed signs of bronchostenosis closely resembling a closed abscess: fever and a distressing cough,

unproductive at times, productive at others. In some with partial occlusions, the symptoms were those, not of a stenosis but of a bronchiectasis: a constant distressing cough with not little but much sputum, often separating into three layers, often blood streaked; at times, as one man described it, white, yellow, green and red. Sometimes, the cough changed: unproductive at first and due to bronchial irritation, it became productive later as abscesses and bronchiectatic cavities obscured the clinical picture of tumor.

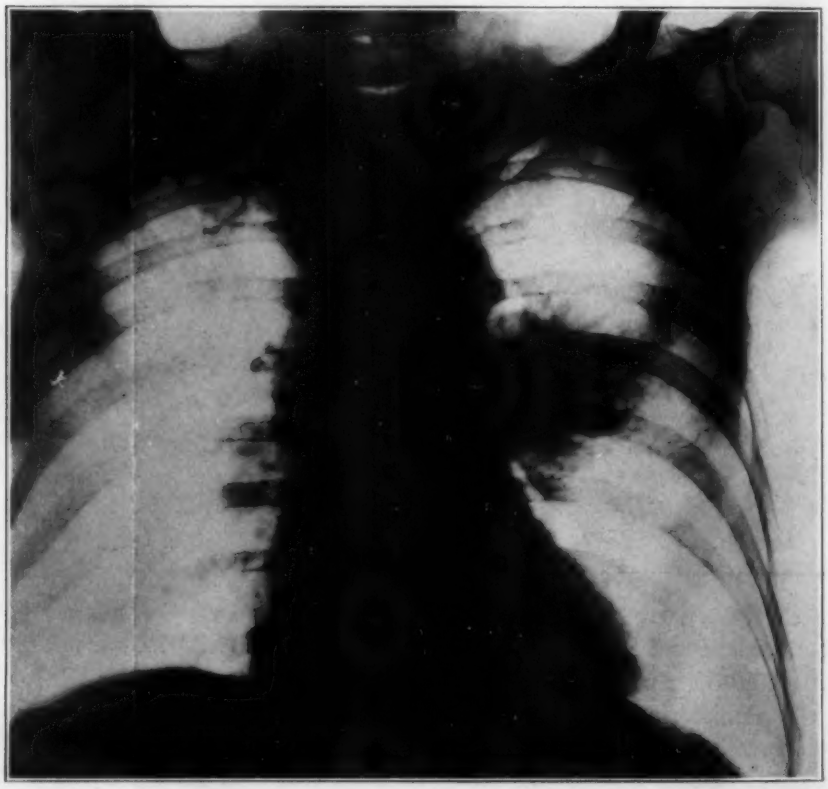

Fig. 1 (Case 3).—Tumor at base of left upper lobe; no chest symptoms; patient entered the hospital for pain in joints.

Three of the patients had localized peripheral growths. Of these, one had no symptoms at all referable to his chest (Fig. 1). He entered the hospital for pain in his joints, and weakness; the tumor was discovered in the course of a fluoroscopic chest examination, undertaken more or less as a matter of routine. One man, whose tumor was attached to the diaphragm, complained only of pain and had no cough and no sputum (Fig. 2). The third suddenly began to spit large

amounts of blood (Figs. 3, 4, 5 and 6). Three other peripheral tumors had formed metastases when they first came under observation.

The appearance of the sputum was determined not by the tumor, but by other circumstances: hemorrhage and concomitant infections. None of our patients coughed up masses of tumor or tumor cells or the currant jelly like material which is said to be characteristic. In one, however, Dr. Scheier's patient, the bronchi were found at necropsy to be filled with a bloody gelatinous sputum much like currant jelly. Examination for elastic fibers was, unfortunately, often neglected. Two of the

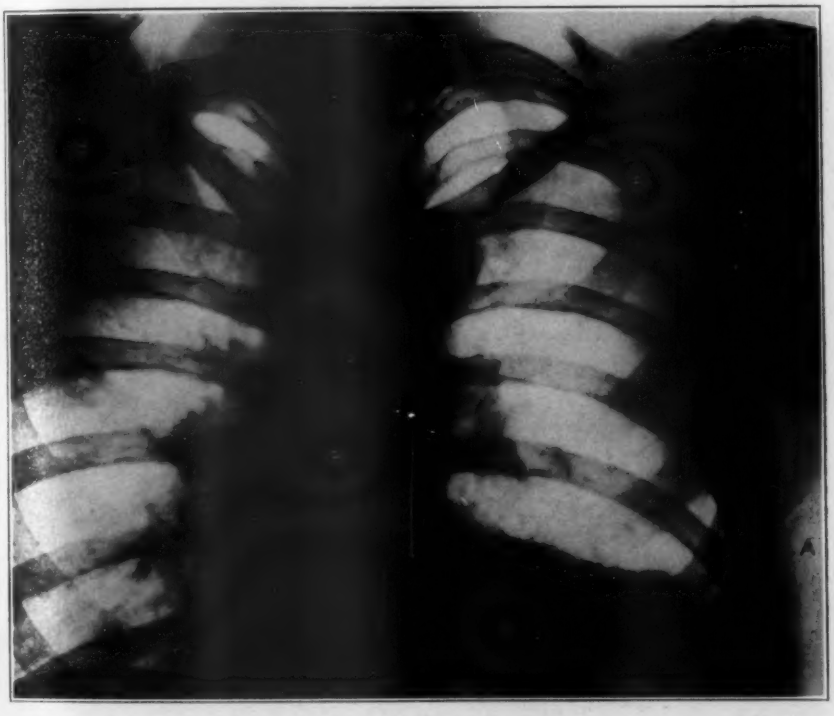

Fig. 2 (Case 5).—A tumor of left base, just above diaphragm; perforation into stomach.

sputums examined showed none. When one inspects the lungs at necropsy, one understands why diagnosis from the sputum will rarely be possible. The tumors seldom project into the bronchi as papillomatous growths; not often will a patient cough up pieces of his tumor, as Barker's [3] did. The tumor masses usually lie peribronchially and under the bronchial mucosa; they do not often slough out into

3. Barker, L. F.: Clinical Medicine, Philadelphia, W. B. Saunders Company, 1922, p. 1.

the bronchial lumen. When they do ulcerate into the bronchi, the sputum is so foul and purulent that any tumor particles that may be coughed up disappear in it. When the sputum is purulent and contains sloughs and elastic fibers, it usually comes, not from the tumor surface, but from an abscess or a bronchiectatic cavity caused by infection of the lung distal to the part of the bronchus which the tumor constricts.

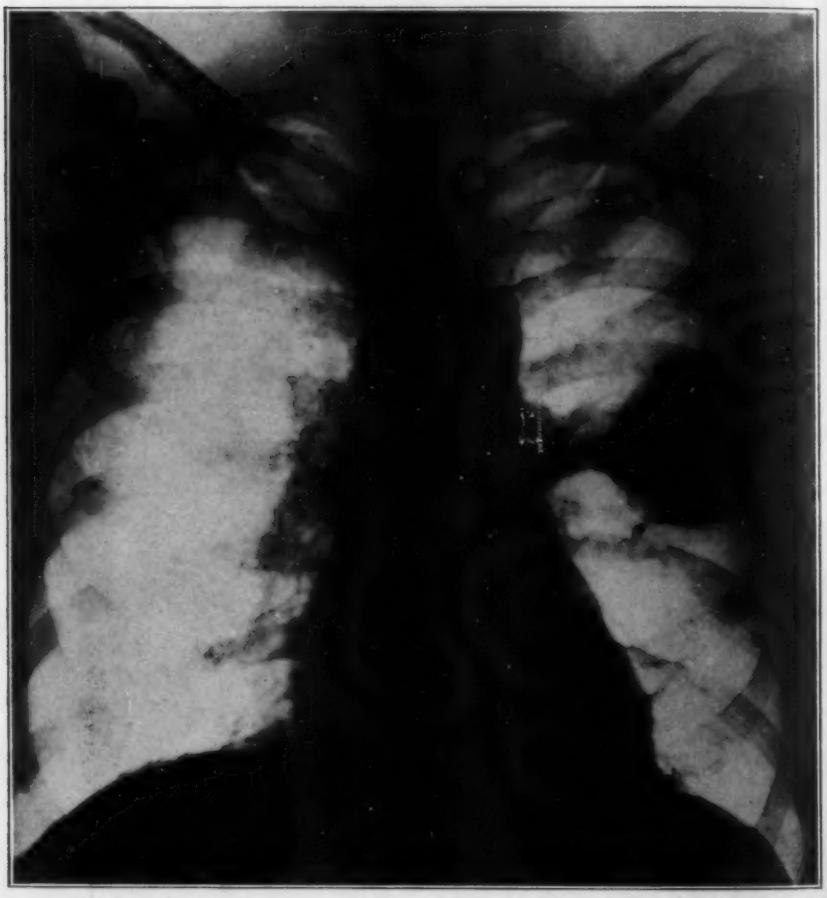

Fig. 3 (Case 6).—Peripheral tumor, Oct. 7, 1921; cough and bloody sputum; no physical signs.

Fever, sweats and chills go with purulent sputum in cases of tumor. They are signs of concomitant infection and suppuration, the formation of abscess cavities and other ulcerations. In one patient, a sudden high fever and violent profuse expectoration signaled the perforation of a tumor of the left lower lobe into the stomach.

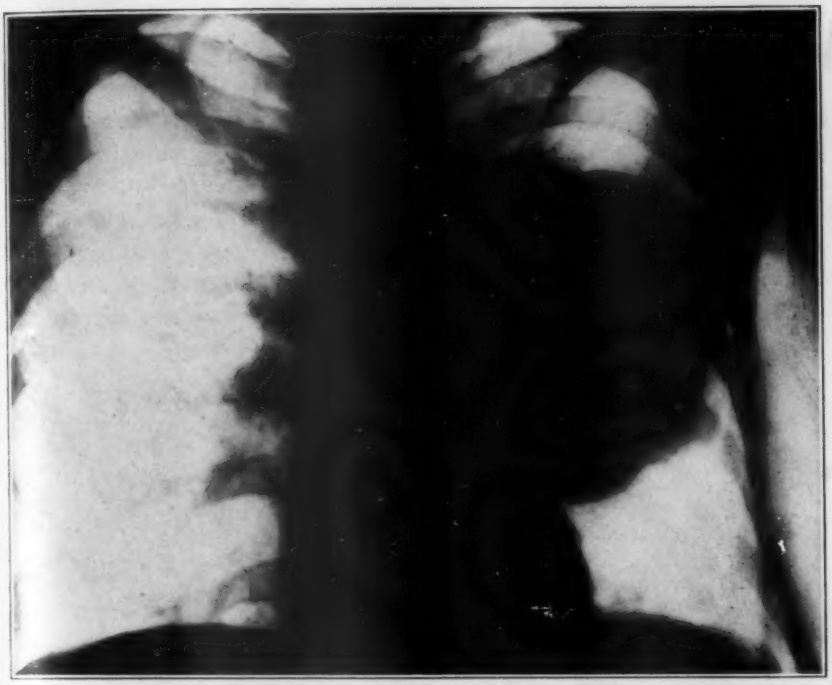

Fig. 4 (Case 6).—Same patient as in Figure 3, thirteen months later, Nov. 27, 1922.

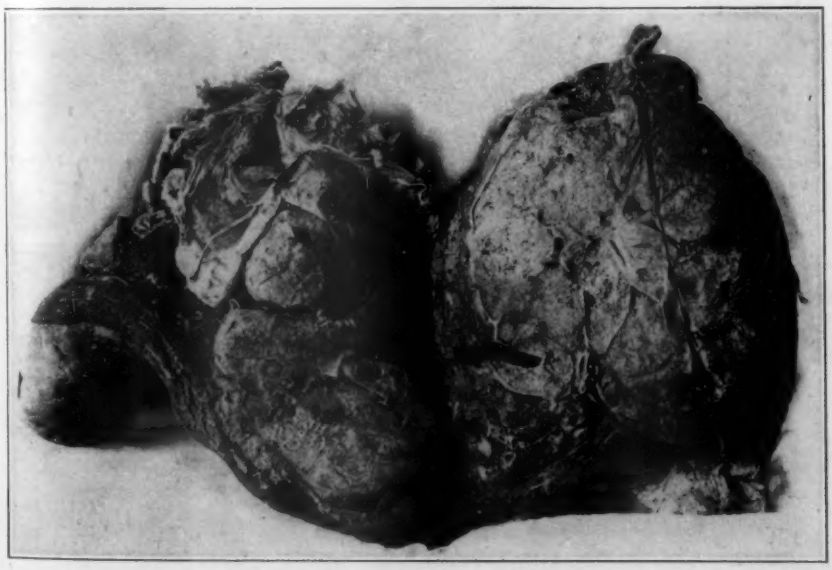

Fig. 5 (Case 6).—Necropsy specimen; lung almost completely substituted by tumor masses; photograph by Dr. Blaisdell.

Once, in Dr. Adler's case, the diagnosis was made by finding shreds composed of tumor cells in the centrifugate of a bloody pleural exudate. Dr. Rusk also established a diagnosis in this way. It seems worth while, therefore, to examine carefully the centrifugate of every bloody pleurisy; embed the sediment in paraffin and section it, if necessary.

Most of the patients had abscess cavities sooner or later. Some of them lay peripheral to the tumor, and were caused by an accumulation of pus in a blocked bronchus; some of them lay at the tumor itself; some of these cavities had gangrenous walls and were traversed by shaggy strands of slough, and by blood vessels which bled more or less violently

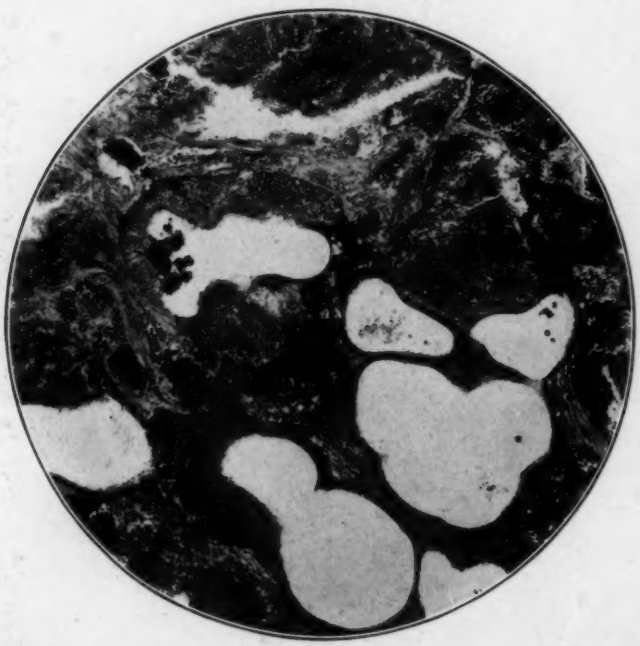

Fig. 6 (Case 6).—Microscopic section; alveoli with central necrosis; small, regular, round and polyhedral cells in the periphery; microphotograph by Dr. Knapp.

as they gave way under the encroachment of the cancer or of the accompanying infection.

Some of the patients lost much weight. The loss could be explained by fever and suppuration, by loss of sleep through pain and cough, or by the presence of other coexistent disease.

Two of our patients had a concomitant phthisis. Tumors which are implanted on old pulmonary tuberculosis, and in which the sputum contains tubercle bacilli, are most difficult to diagnose. The temptation is great, once tubercle bacilli have been demonstrated, to look on the

diagnosis as settled and to let the laboratory get the better of clinical sense. Continual agonizing pain and incompatibility of the roentgenogram with a diagnosis of phthisis, especially the roentgenographic demonstration of a massive consolidation at the hilum or of an entire lobe should awaken suspicion of an accompanying neoplasm. Drs. Randolph and Rusk had the satisfaction of recording the correct clinical diagnosis several times.

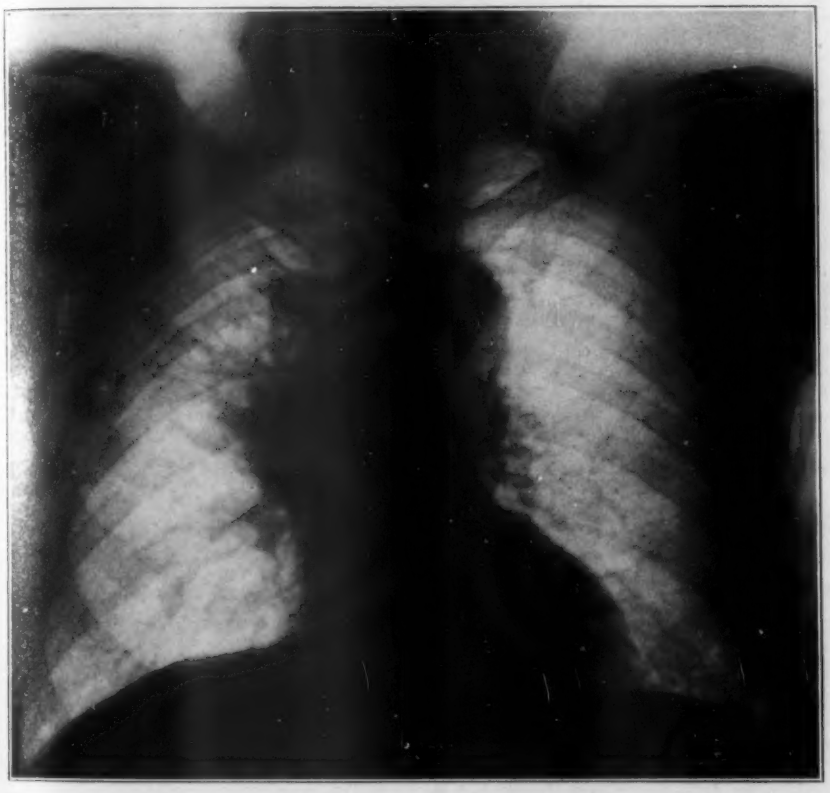

Fig. 7 (Case 12).—Left hilum shadow interpreted as aneurysm; dyspnea, bloody sputum; necropsy disclosed large tumor.

The physical signs in lung cancer were confusing. Obstructing tumors gave signs of either a bronchostenosis or accompanying bronchiectases and abscesses. They lessened respiratory mobility, produced dulness, and changed the breath sounds. Central parenchymatous tumors hid themselves completely from inspection, percussion or auscultation. This is unfortunate. The removable tumors give no signs of their presence; it is the stricturating ones situated near the hilum, whose location makes them inoperable from the beginning, or the complicated

ones with secondary bronchiectases and abscesses, that we can recognize clinically. An unpromising outlook.

The greatest help we had was from the roentgen ray. It was this, and this alone, that revealed the three tumors of the series which, at some time in their course, at least, were operable.

The roentgen ray was a strong ally, but a treacherous one. Four times it led us astray. Three times it led us to diagnose an aortic aneurysm with bronchial stenosis where no aneurysm was found, and

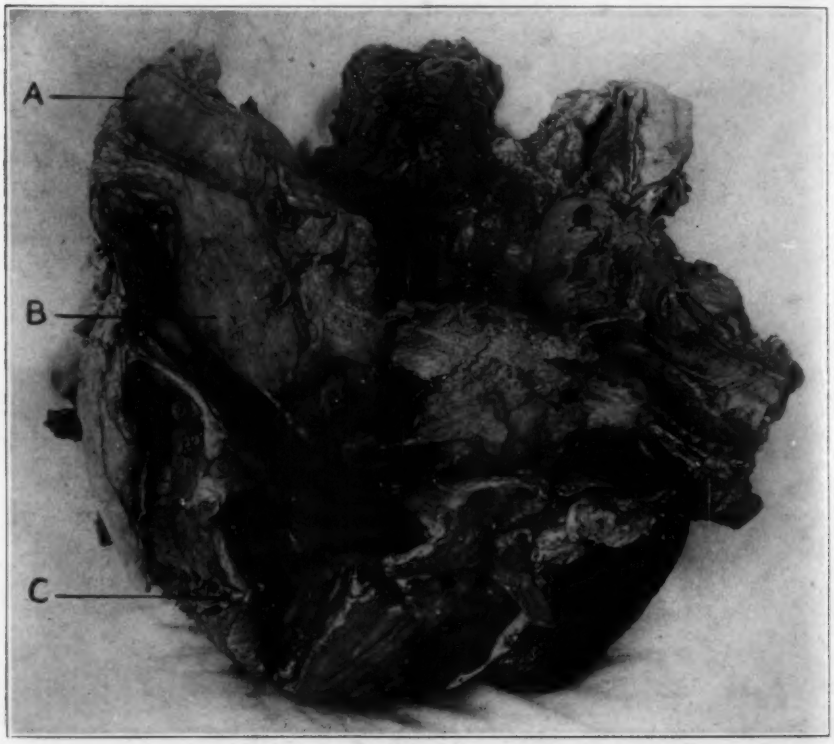

Fig. 8 (Case 12).—Necropsy specimen; massive cancerous infiltrate of mediastinum and pericardium; *A*, trachea; *B*, mediastinal infiltrate; *C*, heart; photograph by Dr. Blaisdell.

it made us doubt whether a tumor visible on the left diaphragm might not be an aneurysmal sac. One of these roentgenologic diagnoses (Case 12, Figs. 7 and 8) is difficult to understand in the light of subsequent evidence. The tumor stands out on the film so characteristically that it is hard to see how it could have been mistaken for an aneurysm.

It is noteworthy that physical and roentgen-ray examination rarely ran parallel. In three patients, we have said, the diagnosis rested on the roentgen-ray examination alone: physical signs

were entirely wanting in two, deceptive in the third. These were the most favorable patients for operation as their tumors lay in the lung parenchyma. Physical examination would never have led to a diagnosis. In ten others, the roentgen-ray examination showed shadows and infiltrates which were due to bronchiectatic suppurations, pleural inflammations and chronic pneumonias. In one, a tumor had been previously

Fig. 9 (Case 4).—*A*, tumor; *B*, abscess cavity; abscess shows fluid level.

diagnosed by the roentgen-ray ; when the patient entered the San Francisco Hospital his symptoms were those of an abscess. Here the roentgenogram showed a typical abscess cavity with a fluid level, but failed at this stage of his disease to disclose a tumor (Figs. 9 and 10). Several times, the roentgenographer reported an infiltrate of the lung extending from the hilum. This was correct as far as it went. Thus, of the central tumors but one was reported specifically as a neoplasm by

the roentgenographer; he reported correctly, however, the three peripheral tumors whose presence escaped the eyes and ears of the clinician.

The clinical diagnosis was correct in ten of the twenty-one cases in which it was noted—a little less than half. In one more, the possibility of a pleural tumor was considered. Bronchiectasis was diagnosed twice; bronchostenosis from aneurysm four times; chronic tuberculosis or pneumoconiosis three times. It is remarkable that the tumor should have been mistaken definitely for an aneurysm four times; in a fifth instance, only an exploratory thoracotomy established the absence

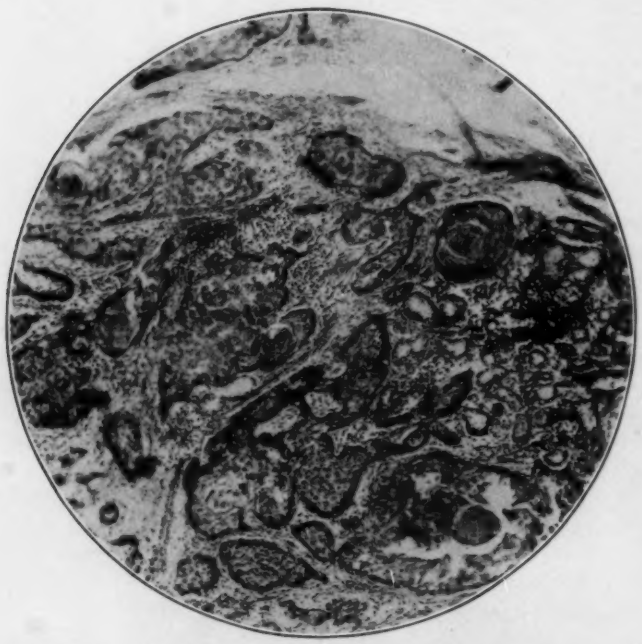

Fig. 10 (Case 4).—Microscopic section; squamous cell carcinoma with horn pearls; microphotograph by Dr. Knapp.

of an aneurysm. This mistake is due to too great respect for the roentgenographer's findings. None of these patients showed clinical signs of aneurysm: murmurs or thrills, pupillary changes or differences between the right and left pulse. The clinicians balked at the diagnosis, even though they did put it down in their records. Once, indeed, Dr. Tupper triumphantly extracted a syringeful of pus from a supposedly aneurysmatic thorax.

One patient was thought to have a tumor who had none. Dr. Evarts Graham and Dr. Bloodgood saw this man, who was sent to me by Dr. H. P. Hill, at the time of the American Medical Association meeting in 1923.

A lawyer, aged 39, previously healthy, had been accepted for life insurance in 1919, 1920 and 1921, but was rejected in 1922 because he had something "wrong with his chest." His physician, Dr. Clark Burnham of Berkeley, and Dr. Voorsanger saw him after this, but found nothing of moment. In April, 1923, he had an attack of fever, cough and joint pain. He recovered from this attack, but, about June 1, 1923, he was suddenly seized with fever, sweats, general malaise and pain in the lower right chest, increased by cough, and relieved by manual pressure against the right ribs. June 9, he began to cough severely but unproductively. The next day, while talking with a friend, he suddenly brought up a quantity of foul sputum and a little blood.

I first saw him, June 14. He had been coughing continuously and was bringing up several ounces of sputum a day.

He was fat and well built, but pale.

Both sides of the chest moved equally on respiration. The lower right chest was dull, especially below the axilla, and the breath sounds were diminished here. A friction rub was heard at times, but there were no râles.

There were no glandular enlargements.

The sputum amounted to from 2 to 5 ounces (59 to 148 c.c.) in twenty-four hours, was purulent, and separated into three layers, the top one apparently containing shaggy fibrinous shreds. It was not foul; it contained small flecks of blood, and was pinkish in color. Neither tubercle bacilli nor elastic fibers could ever be found in it.

The temperature varied from 98 to 100.4 F., the pulse, from 80 to 100, and the respirations were about 20.

The white cells were 14,000 per cubic mm.; the hemoglobin was 75 per cent., and the red blood cells were 4,000,000.

Roentgenograms disclosed a round shadow, without a fluid level, over the right diaphragm, lying rather toward the back. This moved over approximately the same distance as the diaphragm, but between it and the diaphragm there was a fairly, but not quite, clear space. There was no tenting or distortion of the diaphragm on breathing, and its right half moved as well as the left. A few chalky nodules were visible in both lungs, otherwise they were clear, except for the shadow mentioned above (Fig. 11).

In view of the continued absence of a fluid level (even after the patient had expectorated for four days), in view of the movable diaphragm and the absence of a roentgenographically demonstrable inflammatory pleural thickening over the affected lobe, I was more inclined to consider the shadow as that of a tumor, possibly carcinoma of the bronchus, than an abscess. Dr. Chamberlain and Dr. Rehfisch agreed to the roentgenologic diagnosis; Dr. Bryan thought abscess more likely. Dr. Graham considered a tumor more likely and advised removal.

June 27, 1923, the patient was operated on under local anesthesia and gas, administered by Dr. Botsford without inflation of the lung. The fifth to the eighth ribs were resected. A tumor about the size of a hen's egg was felt in the right lower lobe, lying rather more toward the front than the back, over the anterior outer quadrant of the diaphragm, to which it was attached by rather firm adhesions. After these were broken with the finger, the tumor was well palpable, lying in the periphery of the lung. It was resected at one stage. It encroached farther on the hilum than was at first apparent, so that a wedge shaped piece of lung, measuring from about 4 to 4½ inches (10 to 11 cm.) from its edge to the apex, was removed. Clamps were left in place at the hilum, and the chest was closed except for the clamps and a tube which was connected to a suction apparatus.

Immediate frozen sections, seen by Dr. Bloodgood, seemed to show carcinoma, apparently squamous cell.

The clamps were removed on the fifth day. A bronchial fistula made its appearance a few days later; for a week, the patient had fever of from 38 to 39 C. After this, convalescence was uneventful except for an abscess of the soft parts. He was discharged with a closed wound, September 16, and has

Fig. 11.—Oblique projection of a round, solid shadow at right base, interpreted as tumor; resection was done; careful microscopic examination failed to reveal tumor; diagnosis: chronic pneumonia surrounding a small central abscess.

been well since, except for two colds lasting a few days each. He has had no sign of a return of his trouble.

More leisurely studies of the extirpated mass showed a central abscess the size of a small olive, with a greenish thick wall and a massive infiltrate

around it. Dr. Ophüls reported that the lung showed a small, irregular abscess surrounded by a thick layer of dense white tissue. Sections showed a thick, fibrous wall covered on the inside with granulation tissue. Air spaces in the adjoining lung were filled with polymorphonuclear leukocytes and large mononuclear cells, some plugged with granulation tissue. The septums were thickened and heavily infiltrated with lymphocytes. In some places, there was a marked proliferation of the epithelium in some of the partly collapsed air spaces. The epithelial cells were quite unusually large, but there was no evidence of malignancy.

In view of this report, and especially in view of the freedom from recurrence, I think that we must consider the diagnosis of tumor as incorrect and accept the one of "chronic abscess."

I do not see, however, how any measure short of an operation could have established a diagnosis. Lengthy observation would surely have been ill advised, whether for tumor or for abscess.

Of the patients with real tumors, four were operated on. In two, an abscess was drained. One of them had had a previous diagnosis made of tumor. At the time of admission to the San Francisco Hospital, he had a large abscess, possibly resulting from the introduction of radium needles into the lung. The previous diagnosis of tumor was open to doubt; after the abscess was drained, the patient improved for a time. Later, he began to bleed from his drainage tube, and died suddenly after a profuse hemorrhage.

In the second man, a diagnosis of bronchiectatic abscess was made. This man also improved temporarily, but several months after operation had a sudden chill, followed by high fever, and died in two days (Fig. 12).

One man's chest was opened with the hope of removing the tumor. Removal proved impossible: the tumor had grown through the diaphragm and was probably attached to the stomach. The patient died thirteen weeks later, following a perforation into the stomach (Fig. 2).

In a fourth patient, diagnosed as bronchiectasis, a bronchiectatic lobe was removed. He died about twelve hours later. Necropsy disclosed a lymphosarcoma, mainly at the hilum.

But two of the tumors were so located and small enough to make removal feasible. One of them was found by chance in a prematurely senile, arteriosclerotic man who entered the hospital for pain in his joints (Fig. 1). It was decided not to attempt operation on this decrepit person. The remaining patient had a tumor of the left upper lobe, which seemed to offer a good prospect for cure; he refused operation and left the hospital. Thirteen months later he returned, much emaciated, with a huge tumor and enormous liver metastases; he died a week after admission.

Permanent cure of cancer of the lung will probably long remain a surgical *tour de force.*

Sauerbruch reports fifteen operations, five of his own, ten by various other surgeons: ten of these patients died soon after operation; two of them were improved and lived for some time; three were cured.

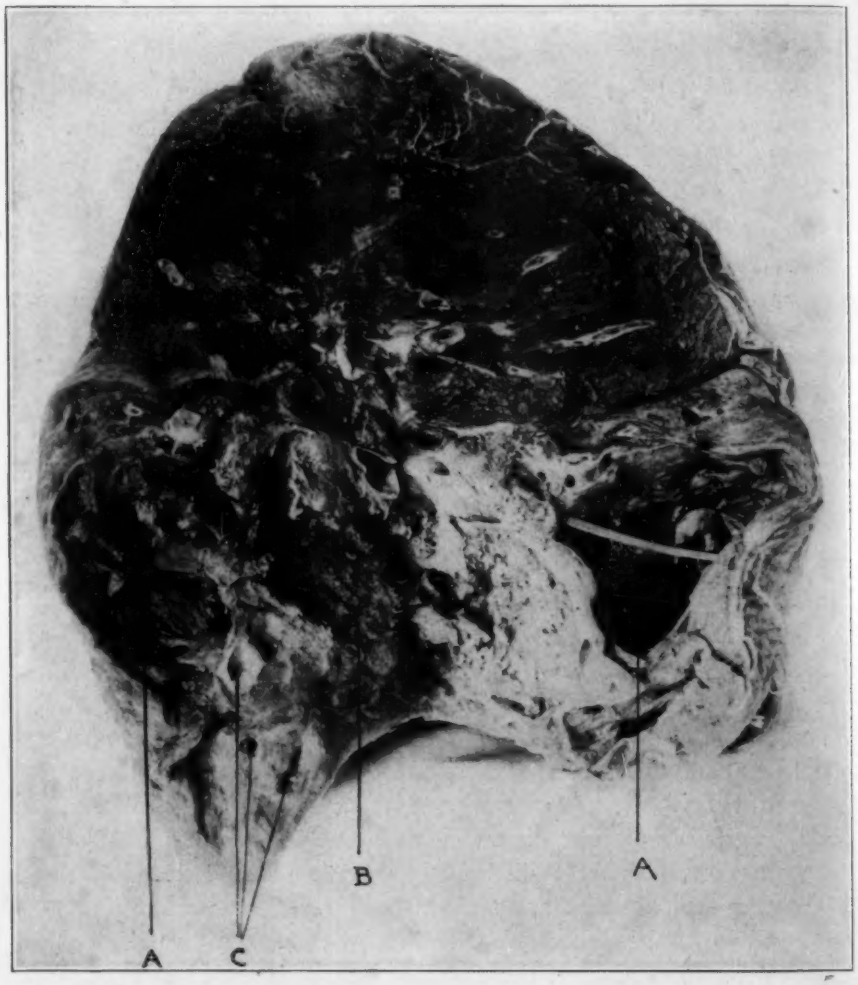

Fig. 12 (Case 9).—Papillary carcinoma of bronchus; large gangrenous cavity lies distal to part of bronchus which tumor occludes; lower lobe is consolidated and threaded by ectatic bronchi; *A*, abscess; *B*, papillary growth projecting into bronchus; *C*, bronchiectases with consolidated lung around them; photograph by Dr. Blaisdell.

Heidenhain resected a bronchiectatic lower lobe, and found in it a carcinomatous nodule the size of a walnut. Sauerbruch extirpated a

Fig. 13 (Case 15).—Section showing bronchial origin of carcinoma; mucosa is wanting over upper half of bronchial lumen; over this area there lie nests of adenocarcinoma; microphotograph by Dr. Knapp.

Fig. 14 (Case 23).—Hilum tumor with large gelatinous nodules; roentgenograms interpreted as aneurysm; photograph by Dr. Blaisdell.

cancer the size of a small fist from the lower lobe; this patient was alive and free of recurrence five years later. In a second patient, he succeeded in resecting a carcinoma of the right lower lobe; the tumor having infiltrated the diaphragm, he removed part of the diaphragm with it. This patient was free of recurrence three years later. I have regretted not having attempted a similar operation on Patient 5 of the table. He was a strongly built man and might have survived.

In all but two of these cases, the diagnosis was established by biopsy or necropsy or both. Eleven of the tumors were of the squamous

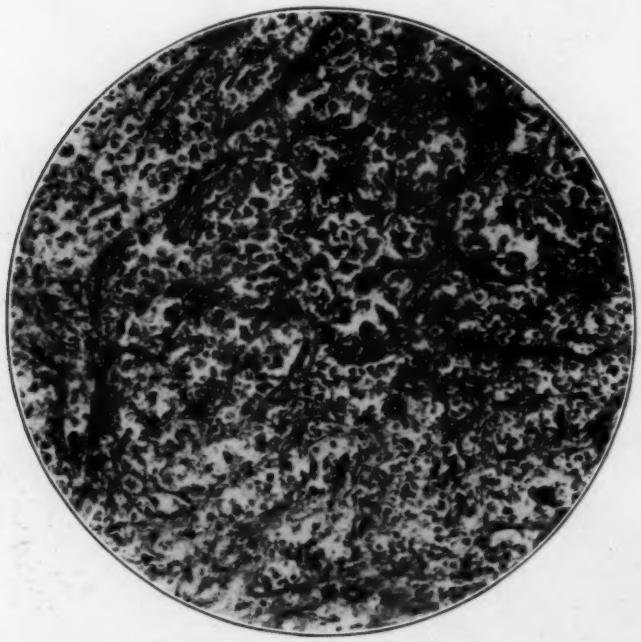

Fig. 15 (Case 25).—Cellular type of tumor; carcinoma? endothelioma? Microphotograph by Dr. Knapp.

cell type of epithelioma; seven were of the medullary type; two were adenocarcinomas; two, lymphosarcomas, and in two no specimen was obtained.

Of the necropsies, eighteen showed the tumor to have originated in a bronchus. Ten of these were of the squamous cell, five of the medullary type; two were adenocarcinomas, one a sarcoma. Three of the tumors, one squamous cell, one medullary cancer, and one sarcoma, seemed to arise from the lung parenchyma rather than from a bronchus; one was a sarcoma, originating near the hilum.

Some writers assert that there are no true cancers of the pulmonary parenchyma; that all lung cancers take their origin from the bronchial epithelium.

A bronchial origin seems proved for many (Fig. 13), the squamous cell epitheliomas and the adenocarcinomas, whose acini are lined with high cylindric epithelium, the counterpart of that lining the bronchial mucosa. Others, more colloidal in type, forming large nodules filled with a mucoid material, with centers that are often necrotic, with cells that have a transparent protoplasm and a large nucleus, seem to resemble the cells found in the bronchial mucous glands which lie scattered throughout the bronchial submucosa (Fig. 14). These are bronchial

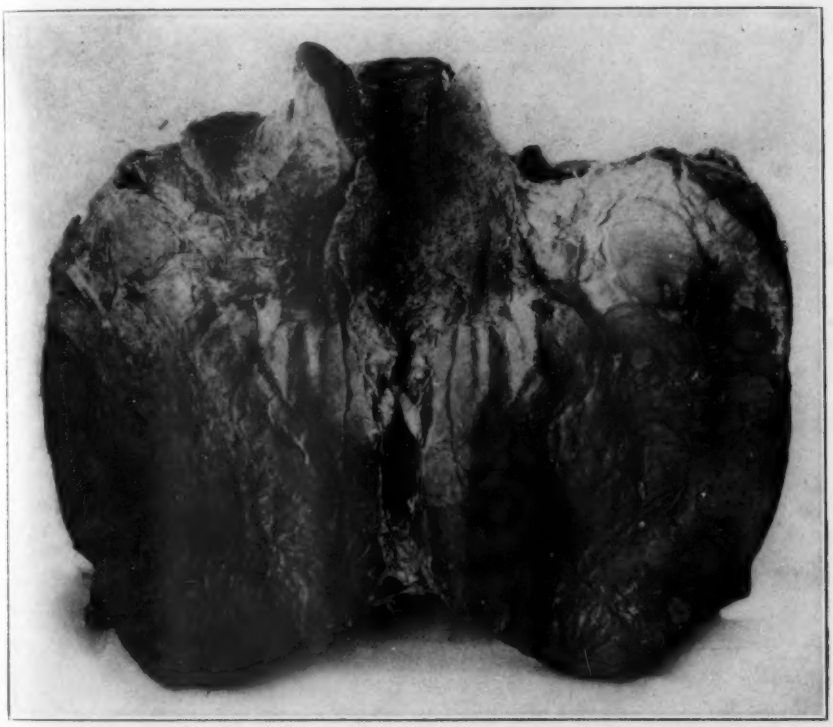

Fig. 16.—Primary sarcoma; lung solidly packed with nodules; photograph by Dr. Blaisdell.

types of tumor; but there are others, notably the medullary carcinomas consisting of small, solidly packed cells, closely resembling endotheliomas both in their structure and in their arrangement, around a fine branching stroma; these and the lymphosarcomas have nothing in their structure that points to a bronchial origin. They may just as well, and probably do, originate in the lung parenchyma. They are rarer, however, than the bronchial cancers.

Different sections of a single tumor often contain cells and cell arrangements of the most varied types: adenocarcinoma in one spot,

squamous cell carcinoma with cornification in another. Several of our sections show this metaplasia: one tumor near the site of its origin had the type of an adenocarcinoma, but at a distance there was found osteoid tissue with spicules of newly formed bone, and marrow spaces filled with fibrous tissue containing solid strands of partly cornified squamous epithelial cells.

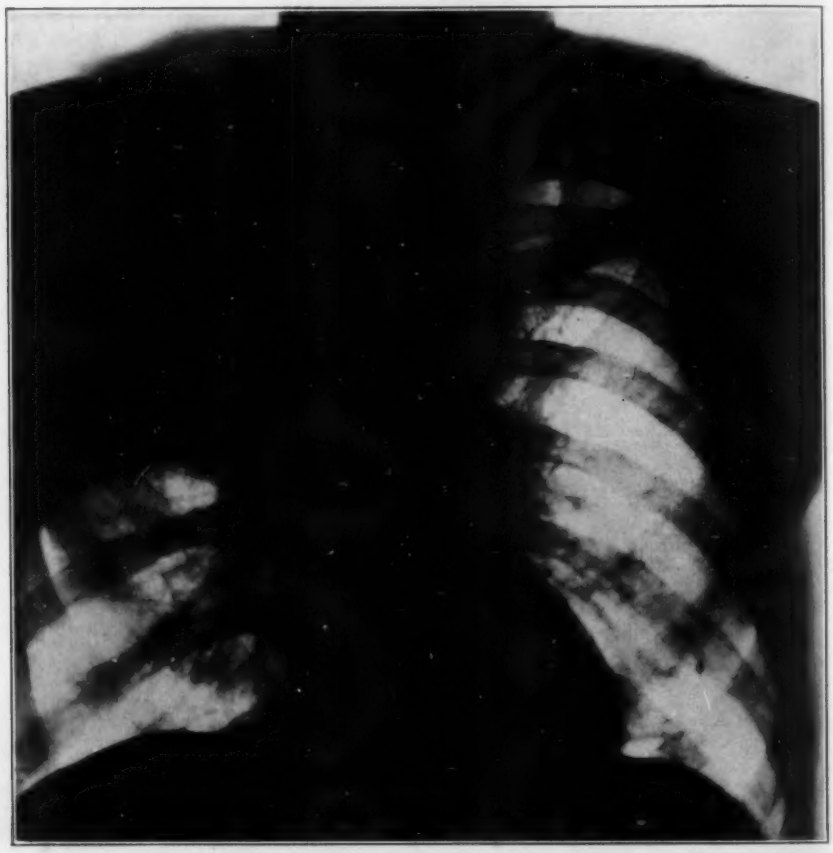

Fig. 17 (Case 11).—Dense consolidation of right upper lobe; patient entered hospital for severe pain in legs; necropsy disclosed primary lymphosarcoma of right upper bronchus.

Sixteen of the tumors affected the left lung, seven the right. Ten lay at the hilum; six affected the upper lobe; six the lower, and one the whole lung.

Supraclavicular glandular metastases were noted four times during life; biopsies of these glands made the diagnosis certain.

At necropsy, the metastases were distributed as follows: in the supraclavicular glands, five; in the peribronchial glands, eleven;

mediastinal, eight, and retroperitoneal, four; in the heart, seven; liver, six; spleen, one; kidney, five; prostate, one; ribs, one; lungs (multiple), four, and in the pancreas, four.

The frequency of heart metastases is noteworthy; no other tumor shows a similar tendency to implant its metastases in the heart muscle. The tumor sometimes reaches the heart by direct continuity, growing through the lymph channels of the hilum, into the sheaths of the great vessels and so through to the heart. The base of the heart, the coronaries, the pulmonary vessels and even the ascending aorta, were twice found imbedded in so massive and hard a growth that it is difficult

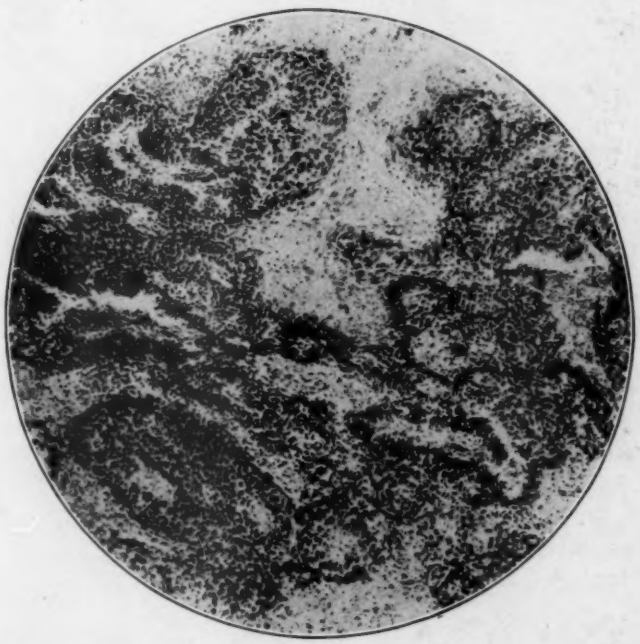

Fig. 18 (Case 2).—Cellular carcinoma without cornification; microphotograph by Dr. Knapp.

to see how the heart could have continued to propel blood through them. Other metastases were deposited in the heart muscle as discrete nodules, probably having entered by the blood stream.

A pleural effusion was noted nine times; pleuritic adhesions, more or less firm, twelve times.

Thrombosis of the iliac veins occurred three times. This thrombosis, when it occurs together with signs of an infiltrate at the pulmonary hilum, may awaken suspicion of a tumor retarding the venous return to the heart.

CONCLUSIONS

Bloody sputum, pain and a dense consolidation should awaken the suspicion of tumor.

Of the twenty-seven cases of primary lung tumor discussed, only two patients were operable when first observed; but five appear to have been operable at any time during the course of their disease.

Primary tumor of the lung occurs with considerable and increasing frequency.

Metastases to the heart occur more frequently than with any other tumor; iliac thrombophlebitis is no rare complication.

135 Stockton Street.

CASE REPORT OF SARCOMA METASTASIS IN THE LUNG SEVENTEEN YEARS AFTER PRIMARY GROWTH

EMIL G. BECK, M.D.
CHICAGO

During the last few years, I have had an opportunity to observe a number of cases of sarcoma of the lung, some of them primary, a number of them metastatic. At this time, I desire to relate only one case of rather unusual interest, and at the same time briefly refer to some of the other cases which have come under my observation. An exhaustive study of these cases will be made when sufficient time has elapsed in the cases in which the patients are still living.

Intrathoracic neoplasms are now more frequently discovered than in the preroentgen-ray period. By means of stereoroentgenograms, we may discover accidently well defined tumor shadows in a chest where no tumor was suspected. A well trained roentgenologist will be able to interpret their meaning. The outline, the shape, size and location of the shadow will enable him to determine, with considerable certainty, as to what type the tumor may be, or whether it is a neoplasm at all. He will be able to differentiate between an abscess, a cyst or an aneurysm, and a neoplasm. A clinical analysis and the physical findings will corroborate or correct his interpretation. A clinical diagnosis of a neoplasm within the chest cavity without a fluoroscopic examination by a competent roentgenologist and a perfect stereoroentgenogram of the chest is rather difficult, and I consider it incomplete. This fact is well shown in the report of Weller, who, in 1913, quoted from the literature ninety cases of carcinoma of the lung, in which series only ten were diagnosed clinically.

Neoplasms in the chest may be malignant or benign. The nonmalignant may be lipoma, fibroma, chondroma or cysts; the malignant, carcinoma, sarcoma, Hodgkin's disease and lymphosarcoma. In the differentiation, we note that the benign tumors grow slowly and do not cause very serious symptoms. Surgically, they are of very little importance.

With the malignant, it is quite the opposite. Their early recognition is the only hope of a cure, and even then the chances are meager. Malignant tumors occurring in the lung may be primary or metastatic. Primary sarcomas in the lung are not as frequent as the metastatic, carcinoma being more frequent than sarcoma. There is very little dif-

ficulty in the diagnosis of the metastatic tumors in the lung because of the history of a primary lesion, which is quite sufficient to draw a correct conclusion. In addition to this, the primary sarcoma is usually a single, well defined tumor, while the metastatic usually consists in tumors ranging in size from a hazelnut to a small orange, often distributed all through the lung tissue, or there is a general dissemination of sarcomatous nodules more uniform in size, ranging from a hazelnut to a walnut, and thickly scattered through the lung tissue. A pleuritic effusion is more common in sarcoma of the lung than in other neoplasms; the exudate is usually serohemorrhagic; but occasionally it is purely sanguineous.

Garré and Quincke [1] state that only thirty cases of primary sarcoma of the lung were published up to that time, but that lymphosarcoma was not quite so rare. The latter originates in the peribronchial glands and usually grows to a large size tumor. This type of sarcoma is very common among miners.

Up to date, I have been unable to find in the literature a single case of primary sarcoma of the lung in which the patient has recovered, but sarcomas originating in the thorax wall or from the pleura have been treated successfully. Cases of chondrosarcoma or a mixed tumor, such as chondromixosarcoma, are even more amenable to treatment.

Kroenlein operated in such a case, in 1883; the patient was a young girl, aged 18, with sarcoma of the thorax wall which had invaded the lung. He excised the tumor, and sutured the lung with catgut. Four years later, he operated on the same patient, resecting a recurrent tumor and a large piece of the lung successfully. Three years later, the girl was still perfectly well. This indicates that even such cases are amenable to treatment. Helfrich went even further; he extirpated a sarcoma of the chest wall and the lower and middle lobe of the lung after he ligated them at the hilum. He also ligated the bronchi by circular purse strings; then he sutured the remaining part of the lung over the stump. The end-result was not so fortunate as that of Kronlein, but the technic employed in this case might serve as a guide in similar cases, and while the final outcome also was fatal, the case is sufficiently instructive to deserve mention at this time.

The question as to whether sarcoma of the lung is an entirely hopeless condition is by no means settled. I am inclined to take a rather optimistic view of this condition; at least, my experience leads me to take that position.

I have in mind a series of cases of sarcoma in other parts of the body, a number of which have stood the test of time, but since they

1. Garré and Quincke, H.: Lung Surgery, 1912.

have not involved the lung, I do not feel that I want to discuss them in a paper treating the phase of metastasis in the lung. Later on, a complete report of these cases may throw an additional light on this important question.

REPORT OF CASE

Sarcoma of wrist, 1907, with amputation of arm; metastasis in lung seventeen years later. A woman, aged 47, a servant of ours, who had been under my observation for seventeen years, when about 30 years old, developed a tumor on the left wrist. My diagnosis was tuberculosis of the metacarpal bones. In my younger days of practice, I did not trust my judgment and consulted Professor Senn,

Fig. 1.—Amputation stump of the arm, 1906.

who diagnosed it sarcoma of the wrist. He pronounced it a very malignant type and advised amputation of the arm above the elbow. The patient refused the amputation, but consented to the removal of two of the carpal bones. Specimens of the tissue were examined by Professor Herzog of Chicago, who diagnosed it as an osteogenic sarcoma. Professor Senn, on this, reiterated his advice to have the amputation done. The patient, being four months pregnant, again refused, and I treated the wrist for a time with roentgen rays, such as were employed at that time, administered with a small gas tube run on a 12 inch (30.4 cm.) coil without any measurements of dosage or distance, mere guess work. There was, as might have been expected, a recurrence so formidable that the woman gladly consented to the amputation; but would not permit removal above the elbow; thus, the arm was removed below the

elbow (Fig. 1). I removed the arm, June 1, 1907. There was a primary union. In due time, she gave birth to a normal child.

There was no recurrence until six years later, when a small growth developed on the scalp in the region of the occipital bone. The tumor was the size of a walnut. I excised the tumor. Microscopic examination proved it to be a spindle cell sarcoma. The wound was then treated without suture, and radium was applied in the open wound, a method which I have now employed for ten years. Since this time (1913), there was no recurrence, so that in the fall of 1921, after a period of fourteen years, I brought the patient before the Chicago Medical Society to demonstrate the permanency of the cure of a sarcoma. Unfortunately, I had no microscopic specimens to prove that this was a true case of sarcoma, and thus I could not blame anyone for doubting it.

Fig. 2.—Recurrence in small tumors on scalp about seventeen years later, 1924.

This suspicion of a mistaken diagnosis was fairly cleared when in February, 1924, the patient again returned complaining of a pain in the chest; loss of weight; that she coughed at night; slept poorly. In February, 1924, four tumors had developed on her scalp, one very near the seat of the tumor removed eleven years before. There was still a very shiny scar in this place. One tumor was about the size of a walnut; the other three were about the size of hazelnuts; they grew quite rapidly. I removed these four growths, and again left the wounds wide open and treated them with radium (Fig. 2). The wounds healed within two months. There is no sign of recurrence as yet. The microscopic sections of the four tumors of the scalp were proved to be fibrosarcoma.

To account for the cough, my suspicion was naturally directed toward the possibly of a metastasis in the lung. To my surprise, I found by stereo-

roentgenogram, that the entire upper lobe of the right lung was clouded by a well defined shadow and two small round tumors in the lower lobe (Fig. 3). We cannot say definitely that the growth in the lung is a sarcoma; but we can think of nothing else in view of the history and the almost typical formation of the small tumors in the lower part of the lung which have appeared since February, 1914.

It is surprising, however, that the patient's general health has considerably improved since last February. She has received deep therapy, by Dr. Orndoff,

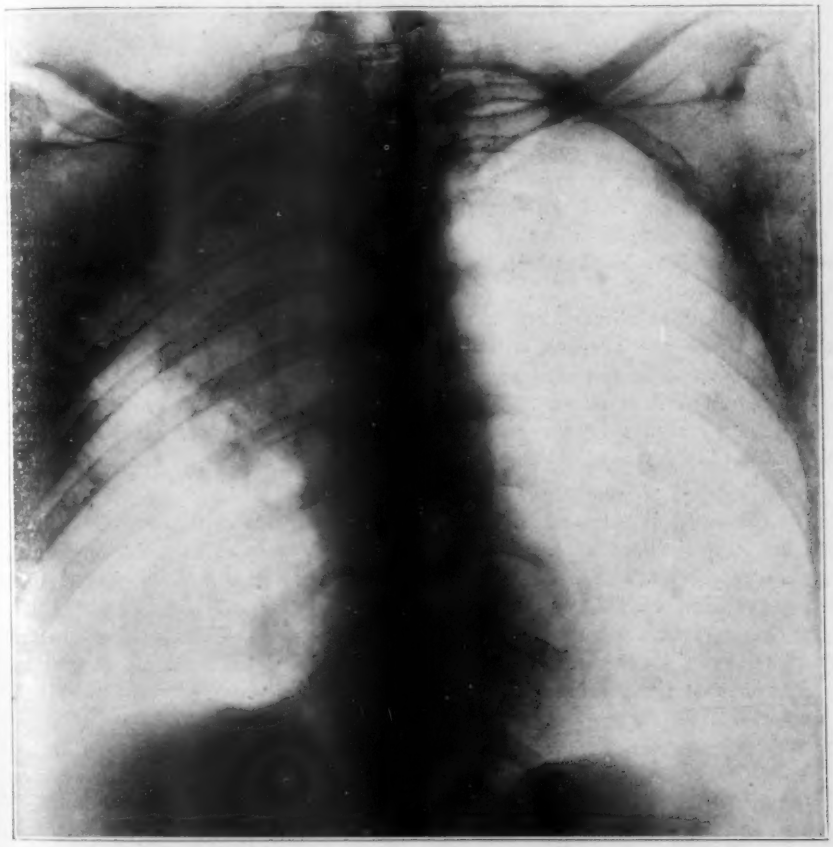

Fig. 3.—Metastasis in the lung, 1924; patient still alive.

and radium applications to the scalp wounds left open after removal of the tumors.

The questions in this case are:

1. Do the shadows in the roentgenograms of the chest represent neoplasms?
2. If so, are they due to sarcoma?
3. If so, is it a metastatic or an independent new growth?

4. Was the growth on the scalp, eleven years before, an independent new growth or a metastatic growth from the wrist sarcoma?

5. In the same way, what relation has the appearance of the four recent growths on the scalp to the original sarcoma of the wrist and the scalp tumor of eleven years before?

DISCUSSION ON THE PAPERS OF DRS. MEYER, ELOESSER AND BECK

DR. WILLIAM H. STEWART, New York: I am afraid from the trend of the papers presented that too much is expected from the roentgen ray. It is a study of shadows, and the element of experience and personality enters very largely into it. One cannot expect to obtain information by this examination beyond the bounds of human ability.

In reference to the localization of abscesses of the lung, simplicity is the best thing. We can, in a great number of cases, accurately localize the lesion by the stereoscopic and the lateral methods. If the case is difficult and one wants an accurate localization, one must use fluoroscopic methods similar to those that Dr. Manges uses in Dr. Jackson's clinic in the localization of foreign bodies.

In locating an abscess, one must have his patient in a position similar to that in which the surgeon will place him when he operates; the phase of respiration must be recorded as well.

I am quite free to admit that the differential diagnosis between a tumor and aneurysm by means of the roentgen-ray examination is often very difficult. We cannot always rely on nonpulsation as indicating tumor; if one succeeds in seeing pulsation it usually indicates aneurysm.

DR. NATHAN W. GREEN, New York: My experience in tumors of the lung is comparatively limited. Of course, we have all seen a great many metastatic tumors of the lung, both carcinoma and sarcoma. It is my impression that the lung seems to filter out of the blood stream the sarcomas, whereas the liver more frequently seems to filter out the carcinomas. I don't know whether that has been the experience of others, but I think it is worthy of consideration.

The metastatic carcinomas, of course, give a definite picture with little circular shadows in the lung, as also do the sarcomas.

The primary carcinomas of the lung, in my experience, have been rather rare and have not been amenable to operative cure. In one case, I have seen a peripheral carcinoma of the lung giving a shadow as if encapsulated. This was attacked by one of our surgeons at St. Luke's, but the effort at removal, I believe, was not successful. I had another case of carcinoma of the bronchus which might have been a primary carcinoma of the bronchus, but the history led rather contrary to that belief. The patient was a man who had had an epithelioma removed from his pharynx some years previously. He was admitted again to the medical ward with the history of bleeding from the bronchus. I made a bronchoscopic examination of him and found an ulcerated tumor at the bifurcation of the lower right bronchus. I transferred him to the Memorial Hospital and there put in radium emanation, but he gradually choked up and died as if he were continuing the progress of his disease.

The bronchoscope helps in the diagnosis of tumors that encroach into the bronchus, but whether we can get them early enough for removal is problematical.

It occurred to me, after hearing Dr. Graham's presentation of his destruction of a lobe in suppuration of the lung that it might be possible to attack these tumors of the lung by a similar method, thereby preventing any disseminating operation.

Dr. Carl Eggers, New York: I am situated similarly to Dr. Green; I have seen a number of patients with tumor of the lung, but I have never seen one that was operable. A young man with a tumor of the right upper lobe with involved supraclavicular glands and metastasis in the liver came under my care a few weeks ago. The pathologic report on one of the excised glands was carcinoma. The primary and secondary tumors grew very rapidly, and led to a fatal outcome in a short time.

I should like to report on two secondary lung tumors which were interesting. Both patients were sent to the hospital with the diagnosis of lung abscess, and both cases proved to be hypernephroma.

The first patient was a young man who was admitted to the service of Dr. Torek at the New York Skin and Cancer Hospital. While under observation for his supposed lung abscess, he developed multiple subcutaneous tumors, the examination of one of which cleared up the diagnosis. He died after a short time. A necropsy was not permitted.

The second patient, Dr. Torek and I saw in the medical service of the Lenox Hill Hospital. An exploratory puncture of the suspected lung abscess showed puslike fluid, which proved to be broken down tumor. The patient was extremely weak and asthenic. The necropsy revealed a bilateral hypernephroma, with lung metastases.

Dr. J. S. Pritchard, Battle Creek, Mich.: From the standpoint of the internist, there are a few points worthy of consideration, particularly in regard to pulmonary metastasis. First, the physical signs; râles, as a rule, are late in coming. They only manifest themselves after some pressure has occurred or there is destruction or disintegration of the pulmonary tissue or the growth itself.

As regards the physical findings of breath sounds, we do not get prolonged expiration in many cases of malignancy in the thorax. We do get, however, in some instances, feeble breath sounds localized. It is an important point to remember that in all cases of suspicion or proved metastasis in any part of the body, it is imperative to have a complete examination of the chest, including the physical examination, the history, the stereoroentgenograms and the lateral roentgenograms, so as to make sure that there is no metastasis present in the pulmonary tissue, as such a condition may exist with very few physical signs or clinical manifestations.

Fever is a common symptom of all pulmonary malignancy or at least of most types. I have observed in the last two and a half years forty-one cases which we diagnosed in my department as pulmonary malignant metastasis.

Dr. Carl A. Hedbloom, Rochester, Minn.: A few months ago, I saw a patient of Colonel Keller's, in Washington, an old man, in whom he had removed the lower lobe of the lung for carcinoma, and at the time I saw him there was practically no carcinomatous tissue left, so far as one could tell, and the man was improving and gaining weight.

Dr. Leo Eloesser, San Francisco: It isn't necessary to emphasize that these are all primary tumors of the lung. Some of them seem to be fairly benign, so that they may be amenable at times to operation.

The diagnosis may be extremely difficult: the roentgen-ray examination often is ambiguous; even with the specimen of lung in hand, it may be difficult

to determine whether one has a chronic fibrous pneumonia or a tumor to deal with. And it may require extensive microscopic study to determine whether epithelial proliferations are innocent ones, such as are regularly found in the smaller bronchi in chronic pneumonia, or whether they are cancerous.

The roentgen-ray examination is of the greatest value in discovering those tumors that make no clinical signs, the peripheral ones. Those that involve the hilum are dominated roentgenographically and clinically by the concomitant suppuration—chronic pneumonia, abscesses and bronchiectasis.

There was but one death in the series attributable to the operation as such.

Dr. Emil G. Beck, Chicago: In the metastatic disseminated tumors of the lung, of course, there is nothing to be done, and I am sorry that some of the gentlemen who have presented papers on radiology haven't told us something of what the prospects are in treating these cases with radiotherapy. About a year ago, with a report, Drs. Evans and Leucutia of Detroit (Deep Roentgen-Ray Therapy of Neoplastic Pulmonary Metastasis, Am. J. Roentgenol. **11**:35 [Jan.] 1924) showed beautiful roentgenograms of lung tumors, before and after treatment, in which the tumors disappeared after treatment. The danger they mentioned in this treatment was resulting fibrosis of the lung, subsequent to the treatment. I think, however, that with some improvement in technic, this drawback may be obviated.

In primary sarcoma, such as I have here shown in a few cases, I see no reason why we should despair. More radical work may bring still better results. Some of our mistakes of the past may be corrected. With additional experience, we may hope for a larger percentage of recoveries.

Some of you remember that, about six or seven years ago, I reported, to the Western Surgical Association, a series of experiments on the cause of carcinoma. We have found torula, which is wild yeast, in the removed growth. This torula is a nucleated yeast cell which grows at extreme temperatures as well as in the incubator. We inoculated guinea-pigs with these cultures, and produced tumors which could not be differentiated from true carcinoma. In 25 per cent. of the animals, we could produce such tumors. What is torula? I inquired at the head of the School of Fermentology of Chicago, and was told that torula, of which there are many varieties, was a yeast fungus found in old beer pumps.

These experiments have never been completed, and the question is still open whether torula plays a part in the etiology of carcinoma.

THE SIGNIFICANCE OF VITAL CAPACITY IN INTRATHORACIC THERAPY*

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A biologic aphorism, no life without breathing, indicates in a general way the importance of respiration. Activities sufficient merely to support life or to realize the utmost physical and mental powers, including defense and repair, are produced by metabolic processes which are dependent primarily on oxidation. This explains why man, although he may survive for weeks without food and for days without water, can exist for only a few minutes deprived of air. It also explains why any reduction in supplies of oxygen to the body, in deliveries of oxygen throughout the body and in utilization of oxygen by the body imposes a corresponding degree of disability.

The many diseases affecting the thorax and its contents, the enormous totals of transient and permanent disabilities and the large number of deaths they cause constitute a serious problem. More effective therapy is needed to provide greater limitations of disability and to reduce mortality.

There is a direct road to this accomplishment. Reduction in vital capacity is so constant a result of thoracic diseases that the extent of this reduction is an accurate measure of the disabilities attributable to them.[1] In order to obtain better methods of treatment, it is essential to know the structures and the functions of the structures that determine vital capacity; how vital capacity is affected by various lesions and consequent malfunctions of these structures, and how therapeutic methods can protect vital capacity against reduction during aggressive

* Experimental and other investigations carried on during and since the war are the basis of this paper, and were aided by grants from the American Red Cross.

1. Estimations of vital capacity are not always the only or even the most dependable sources of information. Frequently, it is impracticable to make them. Occasionally, they can lead to erroneous interpretations. As will be made clear later, vital capacity is influenced by the circulation. An individual may have an apparently competent circulation while at rest and show at such times an approximately normal vital capacity; yet that same individual, if the reserve cardiac energy were limited, would show a material reduction in vital capacity after exercise which would be less of a tax on the myocardium than many types of diseases or operations. Other examples need not be cited. This suffices to emphasize the fact that vital capacity readings, like other single sources of clinical information, are reliable when intelligently interpreted in conjunction with all other evidence.

phases of diseases and facilitate its rehabilitation during regressive phases.

Four distinct phases are notable in completed respiration: (1) ventilation of intrapulmonary air effected by breathing; (2) external respiration, the interchange of gases between intrapulmonary air and intravascular blood through alveolar and capillary walls; (3) transportation of aerated blood to somatic cells and of blood needing aeration to lungs by a coordination of the pulmonary and systemic circulations, and (4) interchange of gases between blood and tissues through capillary walls and cell surfaces or internal respiration. Although most essential, internal respiration is germane to this subject only as it is influenced by the other steps in complete respiration.

Clinical interest centers in the finer workings of the mechanisms of breathing, of circulation and particularly of external respiration because of its direct relationships to vital capacity and to life itself. External respiration not only accomplishes aeration of blood throughout life, no matter how great or how little are the demands for oxygen but this accomplishment has to be relatively complete at any or all intervals. In order to simplify the immediate presentation, let it be supposed that the walls of all of the alveoli are fused into one sheet of epithelium against which is applied a similar sheet composed of the pulmonary capillary endothelium. Suppose, further, that the intrapulmonary air is in an even layer on the epithelial surface and the intrapulmonary blood is flowing in another even layer on the endothelial surface.

Conditions being normal, the oxygen in the layer of air suffices to aerate homogeneously and completely the layer of blood as it passes over the endothelial surface. Should the amount of blood be increased, either the area of the sheet of endothelium must be enlarged or the layer of blood must become thicker. If the layer of blood becomes too thick, it will neither be aerated homogeneously nor completely. On the other side, if the intrapulmonary air be in too thick a layer, because the area of epithelium is too limited, aeration is likewise defective. The states of defective aeration, anoxemia and cyanosis are abnormal. Therefore, it is presumable that there is a natural control which correlates the volume of ventilated air and the area of alveolar epithelium (degree of inflation) with the expanse of endothelium (cross section and length of capillaries) and the amount of blood (unit volume). Moreover, since the demand for oxygen comes from the tissues and is manifested by increments of carbon dioxid in the blood, it is likely that the unit volume of blood in the pulmonary capillaries exerts a telling influence on the three other variables.

A further step in presenting the problem is a supposition that the sheet of alveolar epithelium has been turned into a single sac communi-

cating with a bronchus, and the layer of blood is confined in loops of contiguous capillaries connected with the pulmonary artery and vein. Conditions affecting each of the many alveoli which constitute the portion of the breathing unit directly concerned in external respiration would then be illustrated by a single large alveolus as seen in the accompanying figure.

Obviously, the first and great commandment is to discover the means whereby the amount of blood requiring aeration establishes conditions suited to this accomplishment.

Knowledge of the structures constituting the apparatuses of breathing and of circulation can alone determine therapeutic principles. It is necessary to know how they operate during periods of rest; what adjustments occur when activities are gradually or abruptly increased and diminished; what adaptations are employed to meet handicaps imposed by disease; what are Nature's methods to increase resistance and to hasten repair; what forms of treatment cooperating with natural methods of adjustment, compensation and adaptation will augment defense most effectively and facilitate functional recovery.

Breathing, external respiration and the deliveries of blood to and from the lungs are effected by extrathoracic and intrathoracic structures which are so intimately associated physically or physiologically or both that they must be considered together.

The extrathoracic portions need not be described in detail. It is only necessary to recognize that the chief control of the distribution of blood in the systemic circulation and the rate of flow in both systemic and pulmonary circuits as well as the rate and depth of breathing efforts is in the central nervous system.

The intrathoracic portions not only provide external respiration but they also are the structures affected by the diseases here considered, and give diagnostic, prognostic and therapeutic indications.

An accurate measure of the efficacy of external respiration is vital capacity, which is usually defined as the amount of air expelled by the most complete expiration after fullest inspiration. This means that when the air cells are distended to physiologic limits, inflation and therefore the total area of alveolar walls are greatest for normal conditions and are thus disposed to aerate equivalent amounts of blood.

Aeration is an exchange of oxygen and carbon dioxid through alveolar and capillary walls. Natural economy has apparently established a means of control whereby the total expanse of alveolar wall as determined by the extent of pulmonary inflation compels corresponding variations in the total expanse of capillary wall and the amounts of intracapillary blood. Grades of pulmonary inflation are actively determined by variations in rate, depth and force of breathing movements,

and, as will appear later, are influenced passively by the unit volumes of blood in the pulmonary capillaries. Length, diameter and, therefore, the contents of pulmonary capillaries can be attributed to three forces since there is no effective vasomotor control of the pulmonary circulation. These forces are the blood pressures in the pulmonary circulation, fluctuations in intrapleural negative pressures and alternating intravascular aspiration and expression caused by elongation and shortening of blood vessels with the inspiration and expiration of each breathing cycle.

In addition to central control by the nervous system, there is a peripheral intrathoracic governor which so coordinates the activities of the breathing and circulatory units as to keep volumes of ventilated air present in the alveoli which will aerate at any and all times those amounts of blood being driven through the capillaries. The governor simultaneously correlates the other two factors, namely, the total area of alveolar epithelium and the entire expanse of capillary endothelium.

Knowledge of the structure and function of the governor or gear which coordinates the two sets of variables, volumes of ventilated air and area of alveolar epithelium, with expanse of capillary endothelium and amounts of blood to be aerated, is of basic importance since it determines how external respiration is affected by disease and can be benefited by treatment.

Moreover, such knowledge answers two other significant questions: Why are reductions in vital capacity so accurate a measure of disabilities? What are the structures and what are their activities that influence vital capacity?

Investigation of the various actions and reactions exhibited by the units concerned in respiration during health and the changes imposed by diseases should answer the question. It is necessary to observe the changes in those units under various conditions.

Normal respiration is possible so long as movements of thoracic parietes, particularly of the diaphragm, are unrestricted; intrapleural negative pressures are undisturbed and pulmonary elasticity is not impaired provided the right heart delivers adequate amounts of good blood in the absence of obstruction due to incompetence of the left heart.

NORMAL RESPIRATION DURING REST

During periods of rest, expenditures of energy and hence demands for oxygen are least. In consequence, the volume of tidal air, that exchanged with each complete breathing cycle, is lowest, as is the tidal blood, the amount driven through the pulmonary capillaries during the same breathing cycle. Aeration of tidal blood of rest is accomplished by the tidal air of rest which produces the lowest level of pulmonary

inflation with minimal expenditures of energy. This is the period of greatest physiologic economy in which the storage of energies exceeds expenditures by the largest margin.

The reserve supply of air stored in lungs during periods of rest is five or six times larger than the tidal air. Approximately, three-fifths of the reserve air is available for sudden physiologic requirements leaving two-fifths to meet urgent pathologic demands. A comparable amount of reserve blood available for similar emergencies is stored, less actively circulating, in larger pulmonary vessels.

Tidal air, approximately three-eighteenths of all the air concerned in breathing, serves to ventilate the intrapulmonary air more than enough to keep its oxygen content effectively high. Although one-third of the tidal air is required to fill the trachea and bronchi, the remaining two-thirds causes a change estimated at three-twentieths in the sizes of the air cells.

Thus, there is an established relationship between the volume of tidal air (three-eighteenths) and variation in sizes of air cells (three-twentieths). Presumably, the same relationship obtains between the amount of tidal blood and the length and cross section of capillaries. All combine to afford the means to aeration of the blood circulating through the pulmonary capillaries.

NORMAL RESPIRATION—ACTIVITIES GRADUALLY VARIED

As activities gradually are increased after a period of rest, the slowly progressing demands for oxygen are met by proportionately higher levels of inflation produced by larger volumes of tidal air and by increased amounts of tidal blood driven by higher pressures.

Relationships between tidal air and alveolar size and between tidal blood and capillary dimensions occurring during rest are but little distorted though produced less and less economically as activities increase.

A level is reached which may be termed optimum whereat the tidal air equals the vital capacity. This is the upper limit of economic expenditures of energy at which extraordinary activities can be prolonged without causing early exhaustion. When this level is unusually high, it gives the remarkable endurance exhibited by exceptional athletes, by superior race horses and by certain individuals in meeting the stresses of disease.

Activities raised above the optimum level initiate compensatory responses and mark uneconomic expenditures of energies which assure exhaustion. In order to supply the oxygen demanded for internal respiration, breathing is more rapid and the heart beats faster to develop higher pressures. Finally, a still higher level is reached which may be

called maximum because, at this level, the utmost powers are realized for the brief period before exhaustion forces reduction in activity.

When the sequence is reversed, the processes are orderly reductions in breathing and cardiac rates and diminutions in volume of tidal air, pulmonary inflation, the amounts of tidal blood and capillary expanse. The relationships between the total areas of alveolar walls and capillary walls are adapted to the blood that requires aeration and the intrapulmonary air, properly ventilated, which supplies the oxygen.

Gradually increased activities provide opportunities for orderly adjustments in the actions of respiratory units and develop the largest total power of which an individual may be possessed. Hence the desirability of a suitable warming-up process before severe contests.

NORMAL RESPIRATION—ACTIVITIES VARIED ABRUPTLY

If a resting individual should suddenly engage in a critical physical contest, the demands for oxygen would jump in a few seconds from lowest to highest limits. The reserves of air and of blood stored in the lungs would be utilized until compensation could be accomplished. It has been estimated that the amount of blood delivered by the pulmonary circulation can be increased abruptly more than tenfold. Or should the individual while driving at the maximum level of action suddenly change to complete rest, the rate of external respiration measured by deliveries of aerated blood would decrease with almost the same rapidity.

The relationships between volume of air and total area of air cell wall and amount of blood and expanse of capillary wall must be preserved with a fair degree of accuracy not merely to effect proper aeration of blood but to continue life. A very few seconds suffice to cause dilatation of the right heart or to produce edema of the lungs. These complications are seen infrequently because of a peripheral means of control that maintains the air cell-capillary balance.

Abrupt changes are not alone less economic than are gradual but also are more dangerous, as they permit less opportunity for orderly adjustments and are operated almost entirely by expenditures of reserve energies or margins of safety.

This also applies to the handicaps imposed by disease. The more gradually induced are the less immeditaely dangerous, and this principle should be recognized in methods of treatment. Changes affecting the relationships involved in aeration of blood should be minimized in extent and in the rapidity in which they are produced.

ADAPTATIONS TO PATHOLOGIC STATES

Vital capacity is a suitable term. It does not occur postmortem. When it is less than the tidal air of rest, life cannot continue. If it is

equal to the tidal air of rest, existence is possible until the relationships effecting external respiration are disturbed by increased demands for oxygen. The excess of vital capacity over the tidal air of rest measures ability to work and the margin of safety.

Observations of natural adaptations to the handicaps of diseased states are as valuable as they disclose the compensatory changes that assure the nearest approach to normal external respiration of which the measure is vital capacity.

Normally, a volume of ventilated air on one side of a sheet of alveolar epithelium of exactly proportionate dimenions is separated by the interposition of another wall of vascular endothelium just as exactly proportionate in area to an equivalent amount of blood needing aeration. Normally, these variables fluctuate synchronously and almost equally. The upper limits are set by the total areas of epithelium and endothelium which can be presented, since the volume of air and blood can be further increased by raising the respiratory and cardiac rates for brief periods.

Limitations in any of the four factors—volume of ventilated air, total expanse of alveolar epithelium, total area of vascular endothelium or unit volume of good blood—reduce the essential function, external respiration, which is manifested by corresponding reductions in vital capacity.

Adaptations strive to maintain the broadest margin of safety or the largest vital capacity. When a part of the alveolar epithelium is incapacitated, the remaining portions are expanded so that the total area available for external respiration may remain as nearly normal as possible. This is compensatory or physiologic emphysema. Likewise, the unit volume of blood is correspondingly increased in the capillaries in contact with hyperfunctioning alveoli. This is compensatory or physiologic hyperemia. Morever, the unit volume of blood is reduced in the capillaries adjacent to the incapacitated alveoli. Similarly, increase or decrease in the unit volume of blood delivered through capillaries is associated with equivalent inflation or deflation of corresponding alveoli. Passive congestion is quite different. The capillaries are engorged, but the unit volume of blood passing through them is reduced. Pulmonary elasticity is restricted, and the total area of alveolar epithelial surface is diminished. Reduced vital capacity here also indicates the limitations in external respiration.

The important connection in maintaining the relationships which determine external respiration lies between the air cells and the capillaries. The interdependence of alveolar inflation and volume of air on one side and of capillary size and amount of blood on the other side is obvious.

Air cells and capillaries are not only intimately associated functionally but closely related physically. Capillary loops, arranged in a meshwork, surround, separate and yet connect alveoli. When inflation increases, capillaries are straightened and elongated; when it decreases, they are shorter and more tortuous; hence, the aspiration and expulsion of blood with inspiration and expiration. Contrarily, if tortuous capillaries are straightened and elongated by increased unit volumes of blood, the walls of corresponding air cells are carried with them and inflation results. Also, if this action, which corresponds to a positive phase in erectile tissue, is reversed, capillary walls are less tense, vessels shorten and their lumens are decreased, air cells contract and some deflation occurs.

This, I believe, is the peripheral control, the intrapulmonary governor, and I call it the air cell-capillary gear.

The existence and functions of an air cell-capillary gear are not generally recognized and are disputed by clinicians as well as by physiologists.[2] The latter demand properly controlled crucial animal experiments for proof that would be acceptable to them. They are unaware that one animal, even though denied the distinction of being included among laboratory species, has been giving conclusive although spontaneous demonstrations for centuries. Clinicians have recognized, in those demonstrations, the signs and symptoms of natural adaptations to intrathoracic diseases which occur in man.

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2. Haldane remarked the need of a governor. "We have no guarantee that even during quite normal breathing the distribution of air in the individual lung alveoli corresponds exactly with the distribution of blood to them. Unless this correspondence is exact some alveoli will receive more air in proportion to their blood supply than others, and, as a consequence, the mixed arterial blood will be a mixture of more or less fully arterialized (aerated) blood with some of the consequences first discovered (anoxemia). It is probable indeed that in some way or other the air supply is proportioned to the blood supply whether by regulation through the muscular coats of the bronchioles or regulation of the blood distribution; but it is also certain that this proportioning is only an approximation" (Respiration, New Haven, Conn., Yale University Press, 1922, p. 137).

Certain is it that all the alveoli are unequally supplied with air. Fluoroscopic observations of normal breathing prove this in the increased inflation of the zone of lung adjacent to the diaphragm during each inspiration. The control of air and blood, as suggested by Haldane, would scarcely afford an approximation in the proportioning. On the other hand the air cell-capillary gear provides exact proportioning for each alveolus though not the same proportioning for all alveoli, else there could be no compensation in health nor adaption in disease. Inequalities in alveolar inflation and in the ventilation of intra-alveolar air would occur even in health, and could explain the mixtures of more or less perfectly aerated blood noted by Haldane.

Animals are divisible, as Miller showed, into those having thin pleurae and those having thick pleurae. Animals with thick pleurae have mediastina which are impervious to air and to water. The blood supplied to lung parenchyma and visceral pleura comes chiefly from the bronchial artery. Animals with thin pleurae have mediastina which are pervious to air and to water. Their lung parenchyma and visceral pleura obtain blood supply more largely from the pulmonary artery. Man is of the thick pleura type. Observations made on the usual laboratory animals (cat, dog and rabbit), which are of the thin pleura type, are not directly applicable to man. Attempts to use such experimental observations directly in explanation of human physiologic and pathologic manifestations have led to confusions which are the bases of most misconceptions.

The evidence for an air cell-capillary gear is direct and indirect.

If the pulmonary artery is ligated or obstructed in man or in other animals with a thick pleura, the portion of the lung supplied is deprived of function. It atrophies, contracts and becomes airless. There is no infarction. If a bronchus is ligated or obstructed and pneumonia is not occasioned, the portion of lung supplied is deprived of function. It atrophies, contracts and becomes airless. There is no infarction.

In man, no portion of lung which has had either its air or pulmonary arterial supply destroyed can be functionally rehabilitated. Obviously, if either part of the air cell-capillary gear in incapacitated, the other part is simultaneously disabled.

If the rate and depth of respirations be increased above normal, the carbon dioxid content of the blood is reduced and apnea results. The unit volumes of blood in the pulmonary capillaries are increased because inflation is increased. Extraordinary aeration results, not because of tissue demands expressed through central nervous system control, but because of the coordinating action in breathing and circulatory units effected through the peripheral governor.

If the rate of cardiac contractions is suddenly increased by emotions, there is a correspondingly increased inflation; respirations are deeper and more rapid. There is a transient sense of air hunger. This is a common experience. Inspirations are involuntarily deeper because a larger unit volume of blood is delivered through pulmonary capillaries at higher pressures and inflation of alveoli is inevitable.

Animal experiments are thus far unsatisfactory. Negative pressure cabinets are required to eliminate complicating factors incidental to disturbed intrapleural negative pressures and to the intratracheal positive pressures usually employed. Pneumothorax or hydrothorax induced in an animal with a thick pleura causes a contralateral compensatory emphysema proportionate to the amount of air or water introduced. The animal will tolerate increasing positive pressures so long as it is

able to develop and to maintain the higher pulmonary arterial pressures required to develop the degree of contralateral compensatory emphysema required for external respiration. If the animal be fatigued previous to the experiment, the limit of tolerance is lowered because cardiac energies are less. If the same experiments are made on animals with a thin pleura, pneumothorax and hydrothorax are soon bilateral; compensation is less possible because intrapleural pressures of both chest cavities vary together and toleration is limited. Further confirmation lies in the fact that as animals with a thick pleura are progressively exhausted, the tolerance of intrapleural positive pressures falls until it equals the intolerance of animals with a thin pleura.

Observations have been made often enough during operations on human beings to furnish sufficiently reliable controls to satisfy all requirements for accuracy. The following examples are pertinent. When chronic pleuritic adhesions are divided before pulmonary elasticity has been permanently destroyed, the underlying lung, relieved of restraint, bulges outward if the patient is in fairly good shape even when no differential pressures are employed. If, during an open thoracotomy performed on a strong patient under positive pressure anesthesia, the positive pressures are reduced or stopped, the mediastinum bulges toward the open side. A focal parenchymatous hemorrhage is surrounded by a halo of emphysema. The focal pressure of a finger on visceral pleura will produce emphysema in the adjacent lung.

Another series of observations are notable after lobectomies. Each lobectomy causes greater compensatory emphysema in the remaining lung. The limit of lung capable of supporting life is that which will provide a vital capacity slightly in excess of the tidal air of rest.

Attempts to observe actions of the air cell-capillary gear in excised lungs are quite futile. Postmortem changes occur rapidly. Blood pressures in the bronchial artery are absent. It is impossible to wash out all the blood from the capillaries. The effects of negative pressures are lost. The amount of force which must be applied to drive water through the pulmonary arteries exceeds the strength of the capillary endothelium and alveolar epithelium. Leakage through the air passage results. Inflation of lung by forcing air into bronchi will not cause the pulmonary artery to aspirate fluid because the capillaries are plugged or collapsed. Injecting water into the pulmonary veins causes no inflation just as might be expected because passive congestion is known to reduce vital capacity.

On the other hand, sufficient air pressure in the pulmonary artery will cause inflation of the lung and aspiration of fluid by the bronchus before the capillary walls are ruptured.

The simplest experimental demonstration is to ligate with suitable precautions in a strong living animal the pulmonary artery of one lung and to observe by roentgen-ray examination the increased compensatory emphysema produced in the contralateral lung.

All the manifestations noted can be explained by the interactions of pulmonary inflation and deflation and of the amounts and pressures of blood in pulmonary capillaries effected through the air cell-capillary

Dr. E. K. Dunham's original model of the air cell-capillary gear. The jar represents a pleural cavity. It contains a rubber bag, an air cell instead of a lung, connected with the middle glass tube instead of a bronchus. The glass tube at the left is connected with a coil of guinea-pig's intestine glued to the air cell to imitate a pulmonary capillary. The tube at the right makes it possible to establish negative or positive (intrapleural) pressures. Suitable variations of air pressures exerted through the three tubes demonstrate the effects of breathing, fluctuations in unit volumes of blood delivered to the pulmonary capillaries and changes in intrapleural pressures.

gear. They are supplemented by the forces of blood in the bronchial arteries which fluctuate with physiologic activities of the lung, since they are under control of the systemic vasomotor mechanism.

Dr. E. K. Dunham closed his valuable career with his splendid service on the U. S. Army Empyema Commission. He also had recognized the air cell-capillary governor and constructed a model to illustrate its actions shown in the accompanying figure. The model has been criticized because the pulmonary capillaries have been represented as end vessels. A moment's consideration will explain how difficult or impossible it would have been to reproduce a complete circulation and how unnecessary. Whether or not some blood passes through the capillaries, a sufficient unit volume of blood would cause them to straighten and to elongate.

The adaptations are few in number. Their effectiveness varies directly with grades of competence or incompetence of the circulatory unit. Hence, there are many variations in extent though none in character of responses.

COLLAPSE

Collapse results when intrapleural negative pressures are neutralized by an open thorax or intrapleural exudates of appropriate amounts. One or both lungs may be affected. The extent of collapse is determined by elastic recoil of the lung opposed by the expansive forces exerted through the pulmonary and bronchial arteries. The lower the blood pressures, the greater the collapse. The greater the collapse, the less reserve air and blood remain in the lung, the less the residual air is aerated, the less blood is in circulation and the more urgent the need for contralateral compensation to protect external respiration. Unfortunately, the greater the need, the less the capacity to develop compensation.

Another important influence is the rapidity of reductions in negative pressures. As already noted, adjustments are most effective when they are induced gradually and thus occasion less unfavorable expenditures of energies.

Observations on the effect of war injuries and surgical wounds of the thorax have proved that a competent individual can survive a wide parietal opening even when suddenly produced, whereas a comparable individual, made incompetent by exhaustion, exposure, starvation, hemorrhage, dehydration and infection, can barely tolerate a small opening. Likewise, it has long been known that a gradually increasing pleuritic exudate is of far less moment than one produced rapidly, and that life is possible when the former exceeds in bulk amounts which are fatal in the latter. The extraordinary dangers of abrupt production of bilateral pneumothorax or the rapid formations of bilateral pleural effusions are well known even to laymen. Graham and Bell, working

with animals of the thin pleura type, in which, unlike man, a unilateral pneumothorax cannot be maintained, found that the evil effects of the rapid bilateral reductions in intrapleural negative pressures could be measured by the size of parietal defects. Other experimental evidence has been presented to indicate that if negative pressures are reduced sufficiently gradually, almost normal inflation of the lung can be maintained when parietal defects are created subsequently.

All of these manifestations can be explained physiologically. Nature attempts to maintain suitable areas of alveolar air and blood requiring aeration in order to protect external respiration on which life depends. Should intrapleural negative pressures be reduced sufficiently gradually in a competent individual, there is reason to believe that redistributions of blood in both circuits and increased blood pressures can by action through the air cell-capillary gear maintain full normal inflation after the negative pressures are neutralized. When negative pressures are so rapidly reduced in the competent that compensatory adjustments cannot be made, or gradually reduced in the incompetent, who are unable to make adjustments, corresponding grades of deflation are caused.

Pulmonary deflation inevitably measures an equivalent reduction in the corresponding pulmonary blood supply. It should be recalled that the pulmonary circulation is not provided with an effective vasomotor control. The blood normally destined to reach an area of deflated lung is delivered to the nearest area in which the intracapillary pressures will permit circulation to occur. Wherever an increased amount of blood is driven through pulmonary capillaries there is produced a corresponding degree of inflation, a compensatory emphysema. Thus, if the area of deflation is small, the compensatory emphysema will appear in the same lobe; if larger, in the same lung; if larger still, in the opposite lung. Compensation is produced by the amounts and pressures of blood delivered and is, therefore, most effective in the virile. Limitations of compensatory pressures can be estimated in the weak by determining reductions in circulatory competence as well as by measuring the deficit in vital capacity.

These are Nature's methods of maintaining the air alveolar capillary blood relationships that underlie external respiration. The effectiveness of the relationships is measured by vital capacity. If it is less than the tidal air of rest, life is impossible; if equal, bare existence is possible, and as it exceeds the tidal air of rest, it measures the margin of safety, the limits of possible physical and mental activities including defense and repair.

EXTERNAL COMPRESSION

Collapse is the deflation caused by the elastic recoil of lung when intraplueral negative pressures are neutralized. The amount of deflation

is controlled by the opposing forces, the blood pressures in the pulmonary and bronchial arteries. External compression is produced when a force exceeding the pressure of an atmosphere acts on a lung from without and produces a grade of deflation corresponding to the preponderance of compressive force from without the lung over the expansive resistance of the blood pressures within the lung. On the release of external compression the lung reexpands to the grade of deflation due to collapse. This is often noted when chronic pleural exudates are divided or removed or when pleural effusions are removed.

Deformity of chest walls, intrathoracic tumors, aneurysms, intrapleural transudates, exudates, spontaneous and artificial pneumothorax, thoracoplasty and intrathoracic operations are causes of compression. An outline of changes produced by the common cause, pleuritic exudates, suffices for all.

Exudates formed in a pleural cavity free from adhesions eventually gravitate. Should they increase, the lung is floated upward so far as its hilum attachments permit. As the level of the fluid rises out of the costophrenic sinuses, the lung is elevated and negative pressures are reduced to zero after which external compression begins. It is exerted first on the supra-adjacent lung. This is exactly contrary to the effects of diaphragmatic contractions which diminish pressures on supra-adjacent lung, and are readily seen, fluoroscopically, in the zone of increased inflation just above a contracting diaphragm.

Pulmonary vessels carrying low pressures are easily compressed; air cells are smaller; and this zone of lung becomes proportionately inactive physiologically so that the bronchial arterial blood supply is also correspondingly reduced by the usual vasomotor responses. The same series of changes, becoming more and more accentuated, develop as the exudate increases. With increasing exudation, the significant adaptive changes become more noticeable.

At first, compression of the pulmonary circulation diverts blood to the nearest margin of lung under less compression and creates there a zone of physiologic emphysema in the same lobe. Gradually, this extends to higher lobes and finally to the contralateral lung. Thus are produced zones of skodaic resonance and the contralateral emphysema long noted with pleuritic effusions.

Should the exudate form rapidly, as it often does in streptococcus infections, patients may succumb in a few hours because of their inability to deliver the requisite blood pressures to continue the adaptive emphysema. Physiologic emphysema, be it recalled, is merely a natural method of developing a sufficiently large area of functioning alveolar epithelium in one part of a lung to compensate for reductions elsewhere, and thus to provide enough external respiration to support life. Again,

the exudate may be produced more gradually, and, though it cause complete external compression of the entire lung on the affected side, the patient is able to survive. Moreover, the more gradual productions of exudates are favorable to the formation of adhesions between visceral and parietal pleurae and thus to localization of the process.

Another point is noteworthy. Intense irritation of visceral pleura tends to produce a subserositis or cortical pneumonitis that may be effective in reducing pulmonary elasticity, which interferes with vital capacity.

There is a parallelism between the adaptive processes in the cerebral and pulmonary circulations neither of which is under direct vasomotor control. Both can compensate for gradually increasing antagonistic pressures remarkably well. Both are lethally incapacitated if the counter pressures rise more rapidly than the patient can develop compensatory rises in blood pressures to offset their effects which are respectively cerebral anemia and impaired external respiration.

INTERNAL COMPRESSION

Internal compression differs from external in that the force is exerted within the lung instead of on it. The common causes are chronic passive congestion, pneumonias, tumors, foreign bodies and parenchymatous hemorrhages.

The effects of internal compression are the same as those arising from external compression. They differ in extent and in distribution. Compensatory emphysema develops about a focus of internal compression by the same air cell-capillary gear. The blood being diverted from a focus of greater to surrounding zones of lesser pressures produces an enveloping layer of hyperemia and consequent emphysema. If the foci of internal compression are sufficiently large and numerous, they can produce a manner of skodaic resonance and even lead to contralateral emphysema.

Should the force exerted by foci of internal compression be sufficient, large branches of pulmonary arteries, bronchial arteries or bronchi can be occluded. The effects of such occlusions will be noted later.

PULMONARY CIRCULATION

Abnormalities in the arterial circulation are gradual occlusions of pulmonary arteries producing equally gradual increments in peripheral resistance, sudden occlusions of larger branches by injuries, ligations and emboli causing abrupt increments in peripheral resistance.

Gradual occlusions or obstructions to arterial circulation occur in chronic external and internal compressions of the lung in pleural effusions, tuberculosis, bronchiectasis and pathologic emphysema. High peripheral resistance leads to hypertension in the pulmonary circulation

by extraordinary exertion of the right heart which results in hypertrophy and diminished reserve power. This explains why individuals so affected are especially sensitive to sudden changes in intrathoracic pressures and why all grades of pulmonary compression should be minimized.

Sudden occlusions or obstructions to main branches of the pulmonary artery are produced by ligation and by emboli. They cause a sudden shunting of considerable volumes of blood into other vessels and an abrupt rise in peripheral resistance. A strong individual tolerates such a change without notable variations in pulse rate or systemic blood pressures. A weakened heart may be incapacitated almost immediately. Permanent occlusion of a pulmonary artery causes atrophy, shrinkage and atelectasis in the lung affected. Ligation has been practiced and is still advocated in treating tuberculosis and bronchiectasis. It is in effect a physiologic lobectomy when the principal artery to a lobe is tied. But it does not cause gangrene or infarction because the parenchyma and visceral pleura are supplied by the bronchial arterial circulation.

Experiments indicate that life is possible after four-fifths of the pulmonary arterial circulation has been destroyed in successive stages, as in repeated lobectomies. Sudden occlusions of much more than three-fifths are incompatible with life.

Two erroneous notions about the effects of pulmonary embolism are prevalent. One is that embolism is the cause of gangrene and infarction of the lung; the other, that deaths from emboli can always be attributed to their size.

Embolism can be an indirect cause of gangrene or infarction if it produces secondarily enough internal compression to occlude a bronchial artery. Usually, gangrene and infarction are occasioned by thrombosis in pneumonia or occur in lungs with circulatory impairments incidental to cardiac lesions or consequent on pleural effusions.

The victims of pulmonary embolism expire or they recover, perhaps to die later of pneumonia. Survivors suffer only from such ill effects as may be attributed to a physiologic lobectomy. Deaths from pulmonary embolism in debilitated patients are often due to emboli sufficiently large to raise peripheral resistance abruptly above the limits of their restricted powers of cardiac compensation. There are, however, many deaths of relatively healthy individuals, as, for example, a few weeks after a simple herniotomy, which fail to be thus explained. Barcroft has suggested that an embolus can be caught close to one of the nerve endings present in the pulmonary arterial wall and may, by irritation, interfere reflexly with normal cardiac impulses.

Operative removal of pulmonary emboli has been recommended and, in one instance, has been accomplished without killing the patient. Removal of emboli could not be sufficiently prompt to obviate the sudden

deaths. It could therefore only be employed to rehabilitate a fraction of the pulmonary circulation or prevent embolic pneumonia. The very patients needing to have a portion of their pulmonary circulation restored are those so debilitated that thoracotomy would be a fatal burden. None could say that any given embolus was going to cause metastatic pneumonia, or that its removal would prevent the pneumonia or would help to reduce the dangers of septicemia. Hence, the removal or attempted removal of a pulmonary embolus is almost without exception a highly dramatic example of surgical malpractice.

Interference with the circulation in the pulmonary veins is due to obstructions caused by incompetence of the left heart or resulting from pulmonary compression. Both add to peripheral resistance in the pulmonary circulation.

If the obstruction develops abruptly, and the use of positive pressure with too great anesthesia is a nice example, acute cardiac dilatation is constant and often fatal. Acute venous obstruction can become so critical that systemic venesection is indicated. Gradually increased obstruction leads to chronic passive congestion with its series of handicaps. Abrupt increments in chronic obstruction can cause edema of the lungs.

BRONCHIAL ARTERIAL CIRCULATION

Bronchial arteries carry six times the pressure of the pulmonary artery, and, as stated, supply the major part of nutrition to lung parenchyma and visceral pleura. Hemorrhage from rupture of the bronchial artery into a bronchus causes exceptional deaths with hemoptysis; into a lung, the diffuse parenchymatous hemorrhagic infiltration called splenization. Obstruction of this blood supply can produce infarction and gangrene. The principal significance is surgical. Splenization is commonly a positive indication to excise the portion of lung affected. Incisions and resections of lung require accurate ligation of severed branches of the bronchial artery which are easily recognized by the spurting of red blood. Usually, it is safest to remove all lung bereft of its bronchial arterial blood supply.

BRONCHIAL AIR CIRCULATION

Obstruction of bronchi deprives the corresponding lung of its supply of air and assures atelectasis because the air present at the time obstruction occurs is rapidly absorbed. Commonly, an obstruction of larger bronchi leads to pneumonia through infection added to the pressure exerted by retained secretions.

The surgical significance is clear. Bronchial defects must be repaired accurately, and, when such repair is impossible, the corresponding lung should be removed.

PNEUMONIAS

The more acute and diffuse the inflammation, the greater and more abrupt is the internal compression and increase in peripheral resistance in the pulmonary circulation. Processes of this type are more apt to be accompanied by restricted movements of the diaphragm on the affected side, which are almost certainly inhibited if pleurisy develops. The danger lies chiefly in the extra load thrown on the right heart in addition to the burdens of myocardial injuries from toxemia. It is particularly desirable to reduce the expenditures of energy by the heart. Benefits of early and repeated aspirations of pleural exudates introduced years ago by Bowditch are too important to be neglected. Occasionally, the diaphragm is but little affected. Paralysis obtained by injecting the phrenic nerve can be helpful. The main obligation is to reduce cardiac labor in order to aid compensation and to increase pulmonary blood supply.

Chronic pneumonia reduces pulmonary elasticity and increases peripheral resistance in the pulmonary circuit.

EMPHYSEMA

Pathologic emphysema means reduced pulmonary elasticity, increased peripheral resistance in the pulmonary circulation and a limited capacity for compensation. It commonly indicates a narrower margin of safety than would be suspected. The right heart has been working for a considerable period against abnormally high peripheral resistance, and its store of reserve energy is subnormal. The bronchial arterial supply is reduced, and the powers of resistance and repair in both parenchyma and visceral pleura are accordingly restricted.

Intrathoracic operations on patients suffering from emphysema are extraordinarily hazardous. Such patients are likewise less able to tolerate pleural effusions and should be protected by early and repeated aspirations, preferably by continuous one-way drainage.

ATELECTASIS

Lung becomes atelectatic when its supplies of air or of blood from the pulmonary artery are stopped. It is physiologically inactive and receives the least blood through the bronchial arteries. Such lung atrophies and is cicatrized, both leading to contraction. Contraction is powerful enough to carry with it adjacent lung so that an area of atrophy, which is but a part of a lobe, may reduce the whole lobe to a small mass puckered about the hilum.

In the performance of intrathoracic operations, this must be considered. Lung that has become permanently atelectatic or is likely to become so is more safely excised as a rule.

Observations of the actions and reactions in the breathing and circulatory units occurring under physiologic and pathologic conditions show that nature attempts to maintain such interrelationships as will assure preservation of the basic function of external respiration with the largest margin of safety therein.

Control of the basic relationships, namely volume of ventilated air, area of alveolar epithelium, expanse of capillary endothelium and unit volume of blood, is in part in the central nervous system, but to a larger extent is vested peripherally in the air cell-capillary gear.

Vital capacity measures the efficacy of the relationships so that, in effect, Nature strives to maintain a high normal vital capacity, a lead that therapy must follow.

Vital capacity is determined in the last analysis by the integrity of the pulmonary circulation affected by and affecting the state of the breathing unit. Preservation and rehabilitation of the pulmonary circulation, the quality, quantity and pressures of the blood delivered by it, are the most important part of intrathoracic therapeutics.

NATURAL DEFENSE REACTIONS

Nature's methods of meeting irritations from injury or disease by increasing resistance and hastening repair through reduced but not inhibited function are more evident and perhaps more significant in the chest than elsewhere.

Irritations affecting thoracic parietes or viscera almost constantly restrict parietal movements. Costal excursions may be more affected than diaphragmatic or vice versa. Both are influenced because there is a common control. This is well illustrated if one side of the diaphragm is paralyzed by blocking the cervical portion of the phrenic nerve when there is an immediate, if transient, limitation in the costal excursions on that side.

The effects of restricted motion are to encourage somatic rest, to conserve energy, to limit dissemination of irritants and to prevent atrophy and hyphemia inevitable with nonuse. They may be noted in parietes, in lung and in pleurae.

Restricted motion in extrapleural parietes subject to irritation is of no especial moment other than assuring the richest blood supply compatible with minimal expenditures of energy.

Restricted motion in the parietes has very definite influence on the lungs. When restricted motion is considerable, as in more intense types of irritations, the diaphragm is more or less relaxed and is prone to be forced by intra-abdominal positive pressures into an unusually high position. Pulmonary excursions are thereby reduced, and more important still the total volume of lung or the grade of inflation is less. It has been found experimentally that a lung in approximately a mean position

between extremes of inflation and deflation produced by inspiration and expiration receives the largest unit volume of blood with the least cardiac effort. Under such conditions the vessels are neither elongated nor tortuous and peripheral resistance is lowest. It has also been found that under these most favorable conditions pleuropulmonary resistance to infection is highest, and the rate of repair and functional rehabilitation is well nigh doubled.

The effects of restricted parietal motion on the visceral and parietal pleurae are significant since pleurisy is commonly the cause and frequently the danger of treatment. The parietal pleura receives its blood from the same vessels that supply the adjacent parietes so it is helped by restricted parietal motion. The visceral pleura is more effective in defense against pleural irritations than the parietal because it has a richer blood supply and a larger expanse. The gravity of serositis in general is determined by the excess rate of production of effusions over the rate of their absorption. Resistance of serous cavities is commensurate with their ability to maintain visceral and parietal reflections of serosae in apposition by absorption of exudates. Absorption from the pleural cavity is notoriously slow and slower still if visceral pleura is incapacitated by compression of exudates. Lung in the mean position between inflation and deflation provides its pleural surface with its richest blood supply. Hence, through diminished parietal motion, Nature protects the welfare of parietes, viscera and pleurae.

Besides the foregoing adaptations in the breathing apparatus, there are responses in the circulatory unit. Satisfactory determinations of rates of flow and pressure of blood within the pulmonary circulation are yet to be made. Blood is forced through the lesser circuit in one-fifth the time under one-sixth the pressure required to drive it through the systemic. Fluctuations in blood pressure occur, perhaps synchronously with variations in systemic pressure, but are less in extent. Presumably, the margin of safety in the right heart is as wide as in the left. However, nothing definite is known of the persistence and effects of pulmonary arterial hypertension.

Shunting of blood from one part of a lung to another or to the contralateral lung is attributable in the main to hydrodynamic influences. Anatomists have found nerve cells indicating possible vasomotor controls, but physiologists have not demonstrated corresponding functions. However, two peculiar reactions occur that may be caused by such influences. Massive collapse of an entire lung has followed injury to the opposite side of the chest. Such lung must be deflated, and deflation can result only from failure of air to enter the bronchi or from interference with delivery of blood through the pulmonary arteries. Massive collapse might be explained by a unilateral temporary occlusion near the bifurcation of the trachea were it not for the second reaction. Sur-

geons who have performed open thoracotomy without the protections from differential pressures have noted a sudden lateral shifting of the mediastinum which has been called fluttering or epilepsy of the mediastinum. This has been found experimentally to be associated with equally sudden variations in intrapleural negative pressures, and, in the absence of tracheal obstruction, was attributed to shunting of blood from one pulmonary artery to the other. Conceivably, such shunting might be explained by alternating kinking and unkinking of the pulmonary arteries, but alternating vasomotor spasms seem more plausible.

TREATMENT

The object of treatment is to protect and to restore the function of external respiration, which is estimated by the vital capacity. The means are to assure the integrity of both the circulatory and the breathing units. Available methods are few.

CIRCULATORY UNIT

Abnormalities occur in the amount and distribution of blood in an organism. If the amount of blood has been reduced by hemorrhage or as the result of increased blood destruction or decreased hematopoiesis, prompt relief is attainable with transfusions. If the amount of blood in circulation is reduced because of exemia or the escape of plasma into the tissues, benefits follow intravenous administration of hypertonic glucose which may be given with and without insulin or with and without gum acacia. Each has its indications.

The importance of overcoming shock or states bordering on shock can be illustrated experimentally. If a robust dog is bled from a femoral artery until the heart stops, and an open thorax is then created promptly, the heart beats again for a considerable interval and more blood escapes from the artery. If, however, a dog in a state of prolonged and profound shock is similarly treated, the heart is not reactivated. In other words, little or no reserve blood is stored in pulmonary vessels during shock. Immediate compensatory or adaptive responses are impossible. Additional demands for oxygen cannot be met because an important cog in the mechanism of external respiration is not working; hence, the need for providing an ample blood supply before attempting an operation and avoiding pulmonary compression, so far as possible, if that operation is intrathoracic.

BREATHING UNIT

Means to counteract lesions in the breathing apparatus are extrapleural and intrapleural.

Extrapleural procedures can restrict parietal movements, increase pulmonary deflation or produce compression.

Lesser degrees of restricted movements of the parietes can be obtained by bandages, swathes and adhesive plaster, particularly if it is remembered that adhesive plaster splints are fully effective only when snugly applied, corset-like, around the entire chest. Frequently, desirable benefits can be secured by further increasing the spontaneous restrictions to motion, particularly if the lung is placed simultaneously in that stage of deflation wherein the blood supply is most favorable. Then, benefits may be provided readily by section of the cervical portion of the phrenic nerve, if permanence is required, or by injecting it with cocain or with appropriate dilutions of alcohol should subsequent regeneration be desirable.

Extrapleural thoracoplastic operations are effective in producing permanent compression to aid in obliteration of cavities, particularly in tuberculosis and some forms of bronchiectasis. Failures of these operations, if patients are properly selected, can be attributed to insufficient costal resections or to having attempted too much at one stage.

The operations are dangerous because the patients are usually weakened by a losing struggle against an affection that is mainly unilateral, and during operation they must lie on the sounder side. A more serious handicap is cardiac incompetence. Increased peripheral resistance in the pulmonary circuit has resulted from the internal compression caused by the lesions. It is usually of long duration and has sufficed to reduce the margin of safety especially in the right heart. Fever, intoxication and anemia add burdens by causing myocardial degeneration. It is obvious why excisions of parts of only a few ribs can so alter distribution of blood in the pulmonary circulation that the greater labor required to effect compensation can lead through progressively increasing tachycardia to lethal myocardial exhaustion.

Rest, digitalis, transfusions and intravenous administrations of glucose can be extremely valuable in surgical preparation. The greatest assurance of safety lies in performing these operations in stages with sufficient intervals to permit orderly readjustments in the circulatory apparatus. One stage too many is preferable to one stage too few. An incompleted operation can provide the dangers and distresses of a finished procedure without affording the benefits.

Intrapleural procedures are called closed when free pleural surfaces are not exposed to the air and open when they are so exposed.

Closed methods are drainage afforded by single or repeated aspirations or by the continuous removal of exudates accomplished by airtight, intercostal tube drains, artificial pneumothorax and thoracotomies performed through preformed adhesions. The same sources of danger are present in all. One is the sudden entrance of air into free pleural space. The other is caused by rapid and forceful operating. Both have the same effect, abrupt changes in intrathoracic pressures which require

immediate readjustments in the pulmonary circulation. Likewise, the same precautions are to be observed. Aspirations and drainage through tubes should be gradual. Artificial pneumothorax should be induced slowly and at repeated sittings so that intrapleural pressures are progressively reduced before positive pressures are created. Accurate hemostasis and gentleness are essential in closed thoracotomies.

Empyema will continue to be an important part of intrathoracic therapy. The best treatment is prevention, which means early diagnosis and immediate aspiration as first advised by Bowditch. Fluoroscopy, essential to both, has been insufficiently practiced and is more generally available since bedside roentgen-ray units have been made easily portable.

Early aspiration, perhaps closed drainage, even if it fail to abort empyema, can minimize its severity, extent and the necessity for rib resection. More important still, it reduces the persistence of diaphragmatic paralysis, which Pryor showed to be so common and Middleton found to be commensurate with reduced vital capacity and degrees of disability. Moreover, pulmonary deflation with its added burdens on the pulmonary circulation are minimized during aggressive phases of pneumonias when a slight shift in the patient's favor can obviate a fatality.

Rib resections impair parietal mobility and are to be avoided whenever possible. On the other hand, it is usually unwise to delay performing this operation after the application of ordinary surgical principles indicates open drainage. Much may be accomplished by the use of surgical solution of chlorinated soda (Dakin's solution) to dissolve fibrin and by employing gentian violet, as advocated by Keller, to disintegrate fibrous tissue.

After-care has been too often neglected. As soon as the acute process permits, breathing exercises are needed to stretch adhesions, to reactivate the diaphragm and to develop compensatory physiologic emphysema in order to maintain a high vital capacity.

Discussion of open methods for treating intrathoracic lesions is limited to consideration of ways of performing thoracotomy in the absence of pleural adhesions. Incisions should be designed to assure permanent air-tight healing, else open pyothorax is inevitable. It is the principal cause of postoperative disabilities and deaths. Differential pressure is required for safety. Analgesia suffices. Both can be obtained by the proper use of gas-oxygen as developed by Gwathmey. It is wise to inject the phrenic nerve with 1 per cent. cocain. The immediate paralysis of the diaphragm makes operation easier and hastens recovery. There is less postoperative discomfort as the effects of cocain last for four or more days. Effusions are more rapidly absorbed because the pulmonary blood supply is richest under these conditions and healing of lung tissue is favored. Operators differ in respect to

drainage. Postoperative pleuritic effusions are constant, commonly of considerable volume, are slowly absorbed and offer a favorable medium for bacterial growth. They cause pulmonary compression and its train of evil influences. Air-tight drainage is possible, is effective and is seldom to be regretted. Omission of drainage can be disastrous. While suturing the wound at the end of a thoracotomy, it is important to obtain air-tight closure with lungs in full inflation and to obtain it in such a manner that the stitches may not cause tension necroses.

After-care, if otherwise it can be called such, includes breathing and body exercises to restore parietal movements, pulmonary elasticity and intrapleural negative pressures as steps in the rehabilitation of external respiration which can be measured by frequent estimations of vital capacity.[3]

SUMMARY

Life and activities are made possible by external respiration which is provided through coordinated actions of the breathing and circulatory units.

Coordinated actions of breathing and circulatory units maintain sufficient volumes of ventilated air in contact with a suitable area of alveolar epithelium to assure such interchange of gases through a similar expanse of capillary endothelium as will aerate equivalent amounts of blood. The interrelationships between the volumes of ventilated air, the area of alveolar epithelium, the expanse of capillary endothelium and the amounts of blood needing aeration must remain constant although they are constantly fluctuating with each breathing cycle and with variations in activities of the individual.

This constancy is assured under normal conditions by an arrangement of an air cell-capillary structure and function whereby fluctuations in degrees of inflation produce equivalent fluctuations in amounts of blood and vice versa.

Under abnormal conditions, the relationship is modified through the air cell-capillary gear so that additional areas of alveolar epithelium and of capillary endothelium are provided to compensate for such as may be temporarily or permanently inactivated.

External respiration in a normal individual is undisturbed whether he is at rest or exercising full mental and physical powers. Disability is nil, and the limits of activities, including defense and repair, are measured by normal vital capacity.

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3. Those desiring proofs for assertions made in the text and citations of authors quoted are referred to Wounds of the Thorax, Oxford Surgery, p. 665, and Effects of Acute and Chronic Pneumothorax, Am. J. M. Sc. **165**:1, 1923.

External respiration in persons affected with incompetent breathing or circulatory units is impaired. This impairment restricts the development of mental and physical powers to the level at which the relationships between ventilated air, areas of alveolar epithelium, expanse of capillary endothelium and amounts of blood can no longer be kept constant. There is disability corresponding to the level. Activities including defense and repair are proportionately restricted, and the restriction is measured by the reduction in vital capacity.

Preservation of vital capacity during the acute phases of diseases affecting the thorax and its contents and restoration of vital capacity thereafter are the therapeutic objectives.

Vital capacity measures external respiration which is largely under control of the air cell-capillary gear. Treatment seeks to renew parietal mobility, to reestablish normal intrapleural negative pressures and to restore pulmonary elasticity, at the same time providing for deliveries of suitable amounts of good blood by the right heart and preventing obstruction to the pulmonary circulation through incompetence of the left heart. The specific aim is to rehabilitate the air cell-capillary gear in order to secure the largest measure of external respiration. Achievement is measured by vital capacity.

CONCLUSION

The principal effects of intrathoracic diseases are the malfunctions due to lesions of heart and lungs. Both heart and lungs are possessed of remarkable powers of compensation and repair but, when seriously affected, cause enormous totals of distress, disability and death. Many lesions of heart and lungs from which spontaneous recovery is impossible can be remedied surgically by procedures already devised.

A wider application of known methods and the introduction of new procedures can be hastened by more exact knowledge of intrathoracic structures and functions.

At present, vital capacity is significant in estimating latent powers including defense and repair, in determining therapeutic procedures and in measuring results of treatment.

Vital capacity is regulated by the air cell-capillary gear, the weaker part of which is the circulatory segment. Progress in intrathoracic surgery depends largely on the realization of more effective measures to promote and to conserve the integrity of the pulmonary circulation.

DISCUSSION

DR. WILLIAM D. ANDRUS, Cincinnati: I am interested in what Dr. Yates brings out because it is an attempt to attack the problems of thoracic surgery from the physiologic point of view. The body is acted on by pneumothorax and other thoracic lesions in certain definite ways and has definite mechanisms

with which it responds. So far as we keep these responsive mechanisms on our side, we will be strengthened in our attack; when we go against them, we have them to combat as well.

Dr. Yates has spoken of the question as to the active distention of the lung through a local increase in the blood supply. That this can vary, that is, that increase in blood content does not always mean increased lung volume is shown by the results of passive congestion in the lung. He has mentioned the work of Drinker and Peabody, showing that compression of the pulmonary vein causing passive congestion of the lung lowers the vital capacity quite appreciably. On the other hand, however, active congestion may actually increase the lung volume. Some work has been done by Ebert and by von Basch which shows that the ability of the lung to distend has a relation to this problem, that is, that in an open thorax active congestion will mean more actual distention of the lung than will be the case in a closed chest in all probability due to the changed pressure relations about the lung.

The vital capacity constitutes, in a sense, the cardiorespiratory reserve, or at least one factor of it may be taken as such, and strengthening this reserve in treating thoracic conditions, just as the building up of the cardiac reserve in the treatment of cardiac conditions, means giving the patient a larger factor of safety.

Dr. Evarts A. Graham, St. Louis: In 1918, I thought we had produced evidence of a rather conclusive nature to show that the explanation of why some persons can stand large openings in the chest and some can stand only small openings, in the absence of adhesions, is due to a difference in vital capacity. I have seen no reason to change my opinion in regard to that statement, made in 1918, and repeated many times since.

Dr. Yates deserves much credit for emphasizing particularly the function of the heart, the importance of a normal function of the heart, or as near as possible normal function of the heart, in maintaining the vital capacity.

There are certain things, however, which his presentation brings up with which I feel I must take issue, for example, the statement that he makes that emphysema is produced by the presence of more blood in the lung capillaries. Dr. Andrus, I thought, was going to cover this point, but I think he didn't quite do so. So far as I know, in passive congestion of the lung, one never finds emphysema. I certainly have never seen emphysema in the lungs in a condition of passive congestion, associated with uncompensated heart lesions, for instance. That is a condition in which you have the blood vessels of the lungs, of the pulmonary circuit at least, filled to distention with blood.

Now, in the second place, this interesting model devised by Dr. Dunham is also subject to some fallacy. This model, which I have had the pleasure of seeing a number of times, is constructed by gluing some guinea-pig intestine, to represent the capillary, onto a bladder. Naturally, when the guinea-pig intestine representing the capillary is distended, it is going to pull out the bladder to which it is stuck, but this represents only a single alveolus and a single capillary. Unfortunately, the model does not represent the state of affairs of adjacent alveoli, and we must remember that the capillaries are between alveoli. Consequently we might infer that if all the capillaries are distended the alveoli would have to be compressed.

Now it seems to me that the only way by which we can really understand this question correctly is to subject it to direct experiment, and, so far as I know, the best way to experiment on it is actually to take a lung and inject a branch of the pulmonary artery, or the pulmonary vein, as the case may

be (I don't see that it makes any difference), and actually see if this injection causes a distention of alveoli or does not cause a distention of alveoli.

Mr. Davis and I have carried out a number of experiments on this point, using normal dog lungs, and in every experiment, both in the case of pneumonic lungs and in normal lungs, we have found a very easily demonstrable reduction of size of the alveoli, brought about by injection of a branch of either the pulmonary artery or of the pulmonary vein to the given lobe.

These results have not yet been published, but they will appear with illustrations in the publication of a Harvey lecture which I gave in February. For that reason, I can not agree with Dr. Yates, unless he has some additional evidence, that emphysema is necessarily due to overdistention of the pulmonary vessels.

Dr. Leo Eloesser, San Francisco: I would agree with Dr. Graham's opinion that fibrosis rather than emphysema results from passive congestion of the lungs—if the congestion lasts long enough.

Recent studies of Lange, Felix and other workers in Sauerbruch's clinic have shown that the diaphragm plays a less important rôle in respiration than we are inclined to give it. A few years ago, I had occasion to see two robust stevedores with broken necks in the wards of the San Francisco Hospital. They were paralyzed from the lower cervical segments downward: their intercostal muscles were paralyzed; they breathed only with their diaphragms. We tested their vital capacity soon after admission, before they developed pneumonia, and found it about from one-sixth to one-eighth of what it should have been. This gave us an idea of the relative respiratory value of the intercostal muscles and the diaphragm.

One shoud test the vital capacity in pulmonary tuberculosis before doing a thoracoplastic collapse. If it is low, one should operate in several stages and do a less extensive collapse than when it is fairly within normal limits.

Dr. Herman Moersch, Rochester, Minn.: The relationship of vital capacity to medical and surgical problems is indeed a very interesting one. As so frequently occurs with tests surrounded with theoretical possibilities, the practical and valuable findings are often forgotten in the mass of the theoretical. It would seem advisable, at this time, to recall to the attention of this audience the experimental work Dr. Graham did, in 1920, demonstrating that one can estimate the size of an opening one can make into the thoracic cavity with safety from the vital capacity readings. It is from findings such as this that vital capacity will be of its greatest value to surgery.

I would like to point out that, in making vital capacity estimations, we should first consider what the normal vital capacity is for a person before making pathologic deductions. There are various conditions that influence vital capacity, as for instance: age, occupation, sex, mobility of the thorax, cardiac efficiency and the like. For example, it would be unfair to expect a man leading a sedentary life to have a vital capacity equal to that of an athlete of similar size; or consider variations in them of equal value. Still, that is what occurs in most vital capacity estimations, and it is only from taking all such influences into consideration that we can expect to derive the most benefit from our findings.

There are two findings that I am sure you have all made, and up to the present time no adequate explanation has been offered for them. The first is the observation of Dr. Meyer, who showed in pneumonia that there is a marked decrease in vital capacity which cannot be accounted for by the consolidation of the lung, and, second, the decrease of vital capacity in effusions in which the

effusion has been withdrawn and the lung apparently has expanded. These are two important findings which deserve further consideration and are mentioned with the hope of stimulating an adequate explanation.

Dr. John L. Yates, Milwaukee: I want to take issue with the man who said that this is a theoretical discussion. It is not. Every physiologic fact has a direct practical bearing.

Dr. Andrus mentioned the effects of passive congestion on vital capacity and referred to work by Drinker and Peabody of Boston. The positive influence of circulation on vital capacity is due to the unobstructed blood delivered through the capillaries from the pulmonary artery and not from the pulmonary vein. When there is cardiac incompetence, the outflow of blood through the pulmonary vein is impeded. Drinker and Peabody have shown that there is consequent distention of intrapulmonary capillaries which interferes with elasticity of lungs and encroaches directly on alveolar space. Consequently, under such conditions, there is a reduction in vital capacity and not an increase. This is proved by the fact that, as cardiac compensation is progressively reestablished, the vital capacity increases correspondingly.

As to the criticism of Dr. Dunham's models, it must be realized that he had to construct terminal vessels because he could not provide a complete circulation. You must remember that the tufts of capillaries lie between and separate air cells, and are more or less directly connected with the air cells, so that inflation of the air cells has got to carry with it a certain elongation of the capillaries, and likewise a certain elongation of the capillaries is bound to carry with it, unless there is obstruction to the influx of air, an increased inflation. We have attempted to perform some experiments similar to those Dr. Graham mentioned, and we think we find the contrary, that if the amount of unobstructed blood delivered to a lung through the pulmonary artery is increased, inflation is increased. It is extremely difficult to imitate natural conditions, even approximately, in excised lungs.

Our observations are not limited to animal experiments but also were made on men in good condition whose chests had been opened widely to repair lung wounds. If lung is compressed manually in an interval when no differential pressure is used, it will inflate itself to some extent when released. Likewise, if a lung compressed by a chronic exudate is decorticated, it tends to expand, to bulge through the places where the limiting adhesions are excised. If this may be explained on any basis except the inflation produced by the pulmonary arterial circulation in compressed lung, it is most desirable to have that explanation forthwith.

A statement was made about overaccentuation of the influence of the diaphragm on vital capacity. First of all, let us credit Dr. Pryor of Buffalo for indicating the frequent restriction in diaphragmatic action following pleurisy, how commonly it persisted and its untoward effects.

We have attempted to determine the late effects of lung injuries by examining soldiers so wounded. A year after these men came back from France, Dr. Middleton was enabled to travel around the country to reexamine them. Some had undergone almost complete lobectomy, some partial lobectomy and some had had small wounds sutured. Middleton found that, if he took the remarkably accurate ratings of total physical disabilities developed by the war risk board, compared them to reductions in vital capacity, and then compared both to the amount of restriction of motion of the diaphragm, the three were very much the same. We believe that we have noted in recoveries after intrathoracic intervention, for whatever cause, that the degree of recovery

of vital capacity indicates the total disability and is commensurate with the degree of reestablishment of the function of the diaphragm. The exceptions are notable in younger patients who are able to develop a very high contralateral compensatory emphysema.

It is probable that the size of an opening in the thoracic parietes that can be tolerated depends on vital capacity. All who treated recent chest wounds in France found that a man in good condition, without differential pressure, could stand a wide opening of his chest with comparatively little apparent distress. On the other hand, a man in poor condition could not tolerate so well, if at all, a much smaller opening. A man with good blood pressures could maintain the lung in adequate compensatory emphysema on the opposite side and thus provide sufficient oxygenation, which would be greatly in excess of the compensatory emphysema maintained by the man with the poor blood pressures, simply because the smaller unit volume and lower pressures of blood delivered to his lungs had permitted a greater collapse of the lung in the affected side and failed to compensate for this handicap by producing a commensurate contralateral emphysema to effect necessary aeration. If Dr. Graham will do one thing, he can convince himself as to the actual existence of this air cell-capillary mechanism. The next time he has a relatively normal lung exposed, if he impresses a finger on the pleura, he will see the compensatory emphysema appear around the area of compression; he will see that compensatory emphysema disappears when compression is released provided positive pressures are properly adjusted.

Incontrovertible proof of the existence of an air cell-capillary mechanism, its control and effects will depend on a series of physiologic experiments. Discussion cannot settle this question.

OBSERVATIONS ON THE CARDIORESPIRATORY PHYSIOLOGY FOLLOWING THE COLLAPSE OF ONE LUNG BY BRONCHIAL LIGATION

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CINCINNATI

Surgical pneumothorax exerts its deleterious effects largely through disturbances which it produces in the normal pressure relations within the thorax. On the same side as the opening in the chest wall, the normal negative pressure becomes obliterated, the lung becomes markedly collapsed and the alteration of the pressure relations is transmitted to the opposite side to a degree depending on the condition of the mediastinum. Thus, widespread disturbances are produced in the cardiorespiratory physiology which may be divided, according to their genesis, into four groups:

1. Those produced by the collapse of the lung on the side which is open.
2. Those due to the effects of the changed pressure relations on the lung of the opposite side.
3. Those due to the mechanical interference with efficient inspiration and expiration of the opening in the chest wall.
4. Those due to the changed pressure relations about the heart and great vessels.

Since the cardiorespiratory mechanism is physiologically a dual one, each of these disturbances affects detrimentally both the circulation and the respiration; to offset which the body mobilizes to its aid certain circulatory and respiratory processes of a compensatory nature.

The study of the fundamental abnormal physiology of pneumothorax, therefore, constitutes a very intricate problem when taken as a whole, since both cause and effect are so complex. However, in the study of the physiology of other organs, such as the stomach, in which a certain degree of complexity has made the subject difficult of investigation in its entirety, much has been gained, both in securing and in interpreting data, by studying the component elements of the subject separately and subsequently coordinating the facts thus acquired. May we not, in a similar way, learn something of the fundamental physiology of pneumothorax by analyzing its effects into their component parts?

With such an idea in mind, we have, in animals, collapsed one lung by bronchial ligation in order to reproduce artificially the condition of closed pneumothorax, and have studied the effects of this condition as an isolated problem. This paper is a summary of these effects.

METHODS

In nine dogs, observations were made on the arterial and venous oxygen content, the arterial and venous carbon dioxid content, the respiratory minute volume and the percentage of carbon dioxid in the expired air. From the last two determinations, the total carbon dioxid output per minute was calculated; and this value divided by the difference between the carbon dioxid content of the venous and arterial blood in volumes per cent. gives the amount of blood in hundred cubic centimeters flowing through the lung per minute. Determinations were also made of the pulse and respiratory rate and, in one animal, of the red blood cell count and the oxygen carrying capacity of the blood.

Samples of arterial blood were obtained from puncture of the femoral artery. Samples of venous blood were obtained by puncture of the right side of the heart with a long needle directed upward and inward through the fourth right interspace close to the sternum. All blood gas determinations were made on the whole blood with the apparatus and technic of Van Slyke.[1] The respiratory minute volume was determined by collecting the expired air used for testing samples for carbon dioxid.

In each animal, a complete series of observations was made before operation to serve as a control for subsequent observations. Ether was administered by the intratracheal insufflation method of Meltzer and Auer at a pressure of from 12 to 15 mm. of mercury. The left side of the chest was opened through an incision in the fifth intercostal space; the lung was reflected anteriorly, and the left primary bronchus was isolated from the surrounding tissues. A double ligature of silk was passed about the bronchus, care being taken to avoid including the bronchial artery in the ligature; the insufflation was interrupted for a few seconds to allow collapse of the lung, and, after its collapse, the ligature was firmly tied. The insufflation was again started, and the total occlusion of the bronchus tested by attempts to distend the lung. The chest was then closed with pericostal sutures, the other lung being strongly distended with air before the last suture was tied and normal respiration reestablished by removing the insufflation tube from the trachea.

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1. Van Slyke, D. D., and Stadie, W. D.: The Determination of the Gases of the Blood, J. Biol. Chem. **49**:1-41 (Nov.) 1921.

This operation required only a few minutes for its performance, and was carried out in each case without mishap. In no instance did we see any sign of air in the other pleural cavity or of mediastinal emphysema.

The foregoing procedure was carried out in six dogs of the series; five were kept for subsequent observation; one was sacrificed immediately. In three animals, in addition to the bronchial ligation, a ligature was loosely placed about the left pulmonary artery and attached to a compression clamp, as described by Drinker and Peabody.[2] The shaft of this instrument was brought out through the posterior angle of the incision, and the tissues were sutured firmly about it.

In these three animals, after closure of the chest, the insufflation was maintained at a somewhat lower pressure throughout the experiment, the expired air being collected by means of a right-angled tracheotomy cannula.

As a result of these procedures, the following situation was created. The left lung was maintained in a state of collapse as in surgical pneumothorax, but through distention of the opposite lung just before completing the closure of the chest the normal intrathoracic pressures were, at least approximately, reestablished, and efficient respiratory movements could be performed. Studies could now be made on the abnormal respiratory physiologic condition produced through the collapse of one lung free from other complicating effects of open pneumothorax; and, indirectly, we could obtain some information as to the conditions obtaining in the nonaerated lung. Series of observations were made on all nine animals within thirty minutes of the end of the operation. In five, further series were made twenty-four hours later; and in two, were continued at varying intervals up to the forty-eighth and fiftieth days. In the three animals in which the compression clamp had been applied, the artery was compressed after the first series was completed, and another series of observations was made at once.

SUMMARY OF RESULTS

1. *Effect on the Blood Carbon Dioxid Content.*—(*a*) Arterial Blood: There occurred an immediate fall of 3.5 per cent. by volume in all nine animals. The primary drop was followed within twenty-four hours by a rise of 2.5 per cent. by volume above normal. Subsequently, there was a gradual return to normal between the seventh and fourteenth day. The immediate fall in the arterial carbon dioxid content is inter-

2. Drinker, C. K.; Peabody, F. W., and Blumgart, H. L.: The Effect of Pulmonary Congestion on the Ventilation of the Lungs, J. Exper. Med. **35**:77 (Jan.) 1922.

preted as being the result of increased ventilation of the lung due to insufflation anesthesia (Curve, Fig. 1).

(*b*) Venous Blood: The curve for the carbon dioxid content of the venous blood almost paralleled that of the arterial, but the content was from 3 to 5.5 per cent. by volume greater than in the arterial blood. These curves are essentially the same as those obtained after pneumectomy.[3]

2. *Effect on the Arterial Oxygen Content.*—Following collapse of the lung there is an immediate average fall in the arterial oxygen content

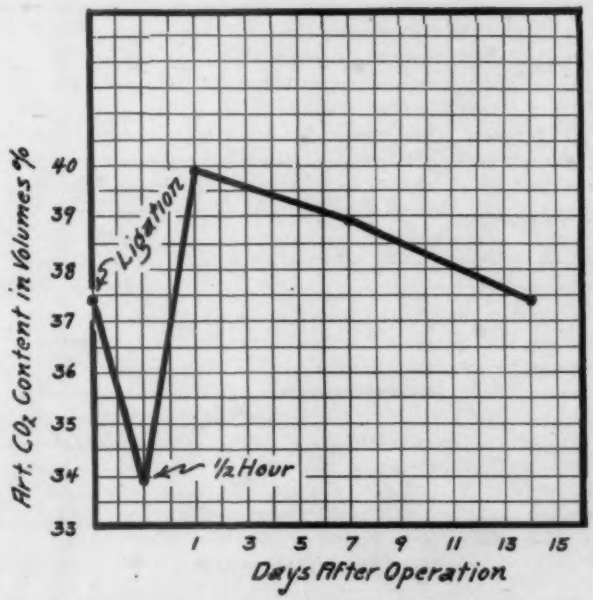

Chart 1.—Arterial carbon dioxid content after ligation of left primary bronchus.

of 5.5 per cent. by volume or a drop of about 30 per cent. below normal. Of three animals repeatedly examined, the oxygen content quickly returned to almost its normal level in one, while, in the other two, it remained about 4 per cent. by volume below normal for forty-eight days, after which it returned practically to its normal level. The oxygen content of the mixed venous blood from the right side of the heart tended to parallel that of the arterial blood and to run from 4 to 7 per

3. Heuer, G. J., and Andrus, W. D. W.: The Alveolar and Blood Gas Changes Following Pneumectomy, Bull. Johns Hopkins Hosp. **33**:130-134 (April) 1922.

cent. by volume below it (Curve 1, Fig. 2). Here, again, we find the picture quite similar to that following pneumectomy.

3. *Effect on the Red Blood Cell Count and the Oxygen Carrying Capacity of the Blood.*—In the single animal in which these determinations were made, there was a sharp rise in both the red cell count and the oxygen carrying capacity of the blood, so that, twenty-four hours after operation, the red blood cell count was increased by 1,650,000 and the oxygen carrying capacity had risen from 21 to 24 per cent. by volume. This corresponds with a calculated increase in hemoglobin of

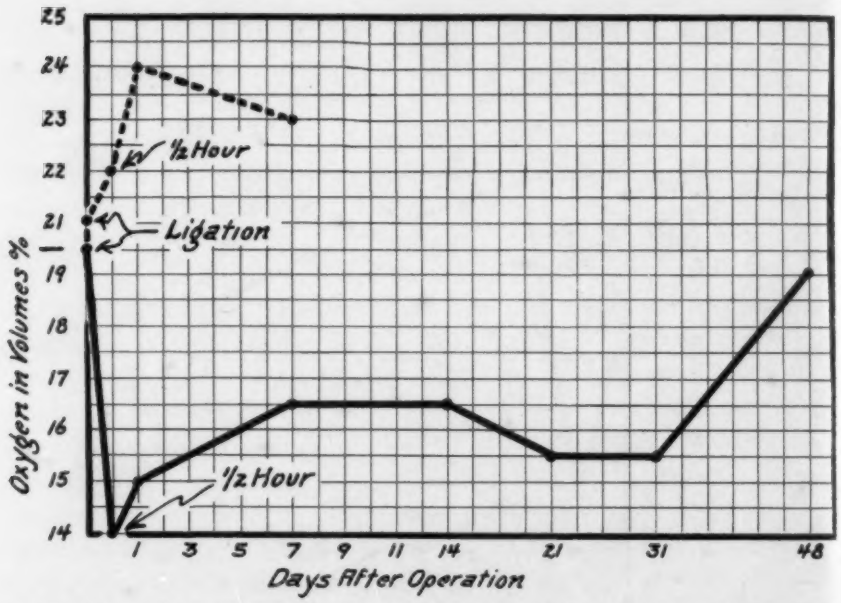

Chart 2.—Effect of ligation of left primary bronchus on the arterial oxygen content and capacity; upper curve: oxygen capacity; lower: arterial oxygen content.

14 + per cent. above normal. Seven days later, the red blood cell count had fallen to 1,200,000 above the preoperative count, and the oxygen capacity was 23 per cent. by volume (Curve 2, Fig. 2).

In our pneumectomy experiments, the rise in these factors was much less rapid and the increase in red cells never so great as in this animal (Fig. 3).

4. *Effect on the Minute Respiratory Volume and Respiratory Rate.*—In the five animals in which this factor was followed, there was an average increase in the minute respiratory volume of about 20 per cent. immediately after operation, i. e., before the effects of the anesthesia had

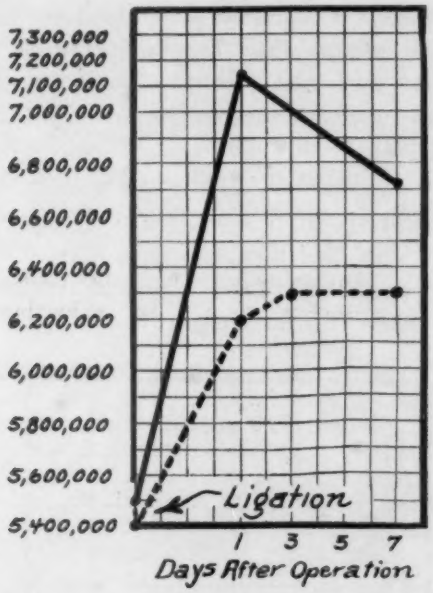

Chart 3.—Comparison of red blood cell count following bronchial ligation and following pneumectomy.

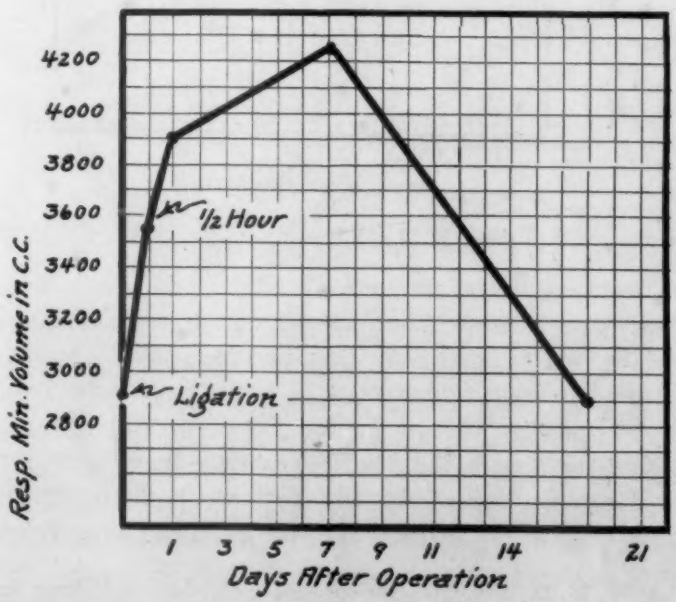

Chart 4.—Effect of ligation on left primary bronchus on respiratory minute volume.

worn off. Twenty-four hours later, the animals were breathing 32 per cent. more air per minute than before operation. In the animals repeatedly examined, there was a 39 per cent. increase seven days after operation, followed by a fall to about normal by the fifteenth to the twentieth day (Fig. 4).

The respiratory rate showed similar changes, being increased by about 20 per cent. immediately after operation and by about 30 per cent. twenty-four hours later, and then falling to a point slightly above the preoperative level by the fourteenth day and remaining at that level during the period covered by these experiments (Fig. 5).

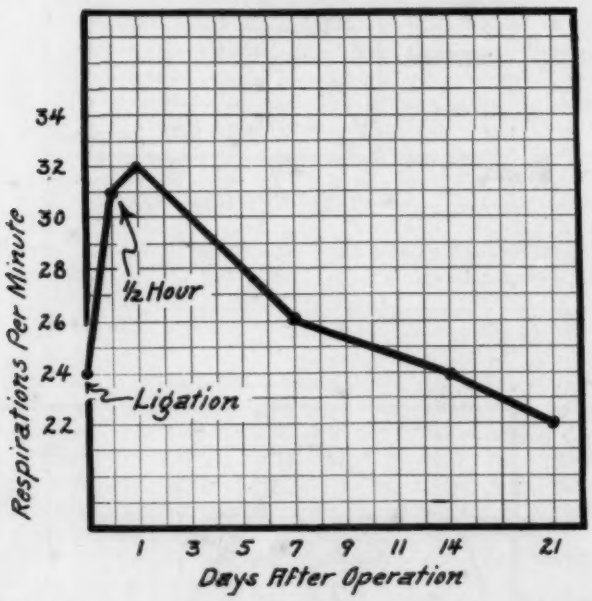

Chart 5.—Effect on respiratory rate of ligation of left primary bronchus.

No direct measurements were made of the total lung volume, but it seems likely that there would be about the same decrease in this following bronchial ligation as there was after pneumectomy. The latter procedure caused an immediate decrease of 42 per cent. in the total volume of air in the lungs.[4]

5. *Effect on the Carbon Dioxid Output.*—Immediately following operation, there is a fall of about 1.1 per cent. in the carbon dioxid in the expired air or about one third of its preoperative value. Twenty-four

4. Andrus, W. D. W.: Observations on the Total Lung Volume and Blood Flow Following Pneumectomy, Bull. Johns Hopkins Hosp. **34**:119-121 (April) 1923.

hours after ligation of the bronchus, this decrease is only about 0.8 per cent., and the preoperative value is restored by about the fourteenth day (Fig. 6).

The total carbon dioxid output per minute undergoes similar changes, being decreased by 30 + per cent. immediately after operation, while, twenty-four hours later, its value is only about 14 per cent. below the preoperative level, which is again reached by about the fourteenth day (Fig. 7).

The considerable decrease immediately after operation is probably due to two factors: First, the lowered blood carbon dioxid from the

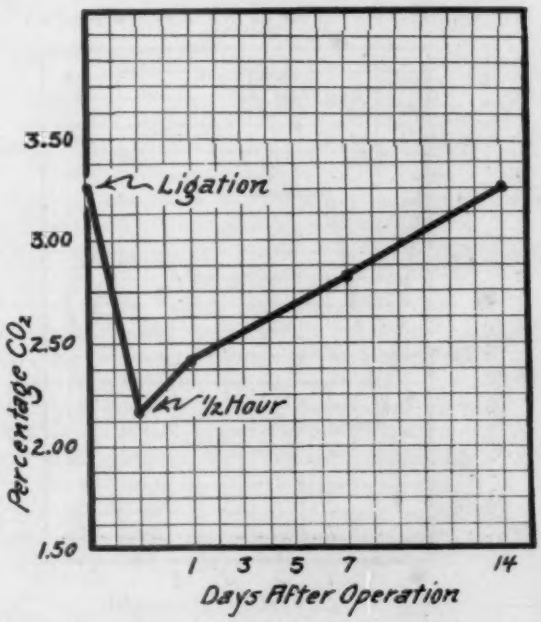

Chart 6.—Effect of ligation of left primary bronchus on the percentage of carbon dioxid in expired air.

washing-out effect of the insufflation anesthesia, and, second, the decreased diffusion space produced by the ligation of the right primary bronchus and the elimination of that lung as an agent in respiratory exchange.

6. *Effect on the Volume of Blood Flowing Through the Lungs per Minute.*—In considering this factor, we shall take up the effects on each lung separately. Previous to operation, we may assume that an equal amount of blood is flowing through each lung, and the amount flowing through the right lung may thus be taken to be one half the average total minute blood volume, or 1,175 c.c. Since we are calculating the

amount of blood flowing through the lung from the carbon dioxid output, our figures refer only to the portions of the lung being aerated and therefore do not refer, except indirectly, to the left lung following its collapse.

Observations made immediately after operation show an average increase in the minute blood flow through the right lung of 655 c.c., or 55 per cent. above the preoperative value. Twenty-four hours after operation, this has increased to an average flow of 1,890 c.c. per minute, or an increase of 60 per cent. In two animals followed further, the right lung continued to transmit from 50 to 90 per cent. more blood than before operation up until the forty-eighth day (Curve 1, Fig. 8).

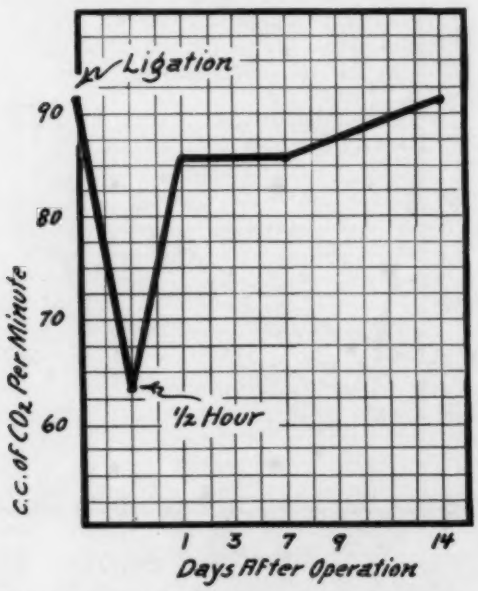

Chart 7.—Effect of ligation of left primary bronchus on total carbon dioxid output per minute.

We are able to estimate the amount of blood flowing through the collapsed lung per minute, and to measure it directly as well. In the first place, we may assume that the decreased blood flow, as measured by the lowered carbon dioxid output following operation, is due to the fact that the blood circulating through the collapsed lung is not giving off its carbon dioxid. Thus, subtracting from the average minute blood flow for both lungs before operation, which was 2,350 c.c., the average minute volume for the right lung after operation, 1,830 c.c., we have the amount of blood circulating through the collapsed lung per minute, which was 530 c.c., or about 23 per cent. of the total minute volume

before operation. If now we obliterate the left pulmonary artery by means of a compression clamp, as was done in three animals, and shunt all the blood through the aerated lung where we are able to measure the flow directly, in the absence of other factors, we should find it thereby increased by the amount which had been flowing through the collapsed lung. This measured about 20 per cent. of the total minute volume before operation in this series. We may be sure then that at least from 20 to 30 per cent. of the cardiac output is circulating through the collapsed lung.

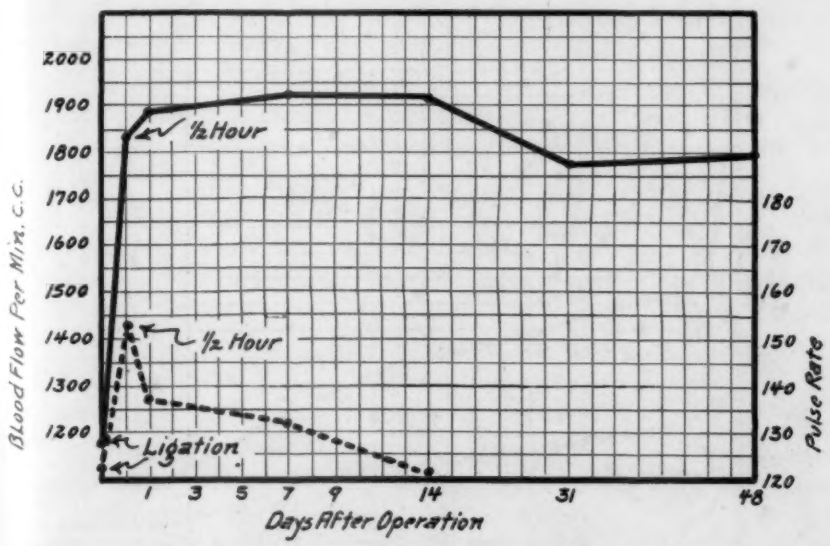

Chart 8.—Effect of ligation of left primary bronchus on the minute blood volume of the right lung and on the pulse rate; upper curve: right lung; lower: pulse rate.

However, the operation produces a rise in the pulse rate which means an increase in the cardiac output, and it has therefore seemed more accurate to calculate the total blood flow per minute from the pulse rate after operation and the pulse volume before operation. By this method, we find a total minute volume for both lungs after operation of 2,680 c.c., an increase of 14 per cent. over the preoperative flow, the average pulse volume before operation being 19.1 c.c., and the rate after operation 141. If now we substract from this the average flow through the right lung after operation, which was 1,830 c.c., we find 850 c.c., or 31 per cent. of the entire cardiac output, flowing through the collapsed lung. As calculated in this way, there is a further decrease twenty-four hours later to 28 per cent. of the entire cardiac output, and this fall continues

up to the thirty-first day, at which time the vessels of the collapsed lung are transmitting but 8 per cent. of the minute output of the right ventricle (Curve 2, Fig. 9).

7. *The Effect on the Pulse Rate.*—Accompanying these changes in the minute volume there are corresponding changes in the pulse rate. Immediately after operation, this is increased by eighteen beats per minute, or about 14 per cent. Twenty-four hours later, the rate is increased only fourteen beats, or 12 per cent., and from then on it

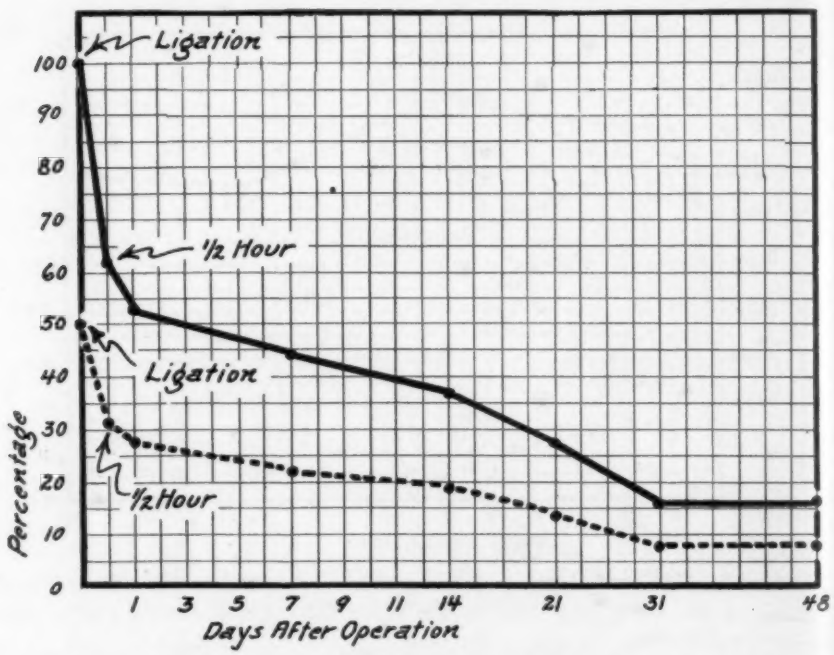

Chart 9.—Effect of ligation of left primary bronchus on circulation through left lung (calculated); upper curve: circulation through left lung as compared to its own circulation before operation; lower: percentage of the total calculated cardiac output flowing through the collapsed (left) lung.

gradually drops, reaching normal about the fourteenth day. Further observations on two animals showed a fall below the preoperative rate in one, while, in the other, the rate was equal to that before operation on the forty-eighth day (Curve 1, Fig. 8).

Pneumectomy produced a similar effect on the pulse rate as observed in our previous series of experiments.

Since we have no means at hand of directly measuring the total cardiac output following bronchial ligation, we cannot follow the pulse

volume directly, but, in the animals in which a compression of the pulmonary artery was carried out following the bronchial ligation, the pulse volume was found to have been increased by about five per cent. by this procedure. In our previous experiments, we found an increased pulse volume following pneumectomy of about 5 per cent.

8. *Anatomic Changes.*—The collapse of one lung by bronchial ligation produces ultimate anatomic changes in the other lung and in the thorax in general similar to those which have been shown to occur following pneumectomy.[5] The space left by the collapse of the lung is obliterated, three factors taking part, the mediastinum being dislocated toward the collapsed lung, the diaphragm on that side becoming somewhat elevated, and the chest wall collapsing to a certain degree.

At necropsy about four and one-half months after ligation of the left primary bronchus, the following conditions were found: The right lung was markedly enlarged and had dislocated the mediastinum considerably to the left. Grossly, it appeared quite normal, and was removed and distended with air at 10 mm. of mercury pressure, and fixed. On microscopic examination, the alveoli were seen to be dilated but unruptured; their lumens were clear and their walls, though thinned out, showed capillaries containing blood. The bronchi appeared normal but seemed slightly dilated, their mucosa being flattened, and branches of the pulmonary artery and vein could be seen filled with blood. Fibrosis, as seen in true emphysema, did not seem to be present.

The left lung appeared shrunken to about from one eighth to one tenth its normal size, bluish red and of liver-like consistency. The ligature was found intact, and no dilatation of the bronchi beyond was discernible. On sectioning with a knife, the cut surface bled but was of definitely increased density. Bronchi and bronchioles were present on the cut surface, but other lung structures were obliterated.

Microscopic section showed the bronchus intact and not dilated, as the mucosa was seen to be in folds. Its lumen was filled with clear mucoid material containing some cellular debris. The accompanying branch of the pulmonary artery and radicles of the pulmonary vein seen elsewhere contained blood. The pleura appeared slightly thickened and beneath this the remainder of the lung was made up of a dense, cellular structure which seemed to be composed of the cells of the alveolar walls with many fibroblasts. Blood cells could be seen scattered between the other elements, but, except for some well marked veins and branches of the pulmonary accompanying the finer bronchi, no real vessels could be made out.

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5. Heuer, G. J., and Dunn, G. R.: Experimental Pneumectomy, Bull. Johns Hopkins Hosp. **21**:31-42 (Feb.) 1920.

A lung removed three days after bronchial ligation appeared of somewhat softer consistency than the later specimens, and, on microscopic section, was seen to be merely atelectatic; the aleveoli were collapsed, but there was no organization. The entire structure appeared less dense and more blood vessels could be seen in this than in the lung examined four and one-half months later.

SUMMARY

Briefly, then, the collapse of the left lung, in dogs, through ligation of the left primary bronchus, produces the following immediate results:

1. The collapsed lung still transmits about 30 per cent. of the calculated total cardiac output.

2. There is a decrease of about 42 per cent. in the total lung volume; this decrease corresponding to that following pneumectomy.

3. These two factors cause a reduction of 30 per cent. in the carbon dioxid of the expired air, an increase in the carbon dioxid content of the arterial and venous blood, and a decrease of 5.5 per cent. by volume (30 per cent.) in arterial oxygen content.

To offset these deleterious effects, simultaneous changes of a compensatory nature occur as follows:

1. The respiratory minute volume and the rate of respiration are increased by about 20 per cent.

2. The pulse rate is increased by about 14 per cent. with a corresponding increase in the total cardiac output.

3. The circulation through the aerated lung is increased by 60 per cent. in volume per minute.

4. Within twenty-four hours there is a marked increase in the red cell count and the oxygen carrying capacity of the blood.

The increased blood carbon dioxid, the decreased carbon dioxid output, and the increase in the pulse rate, cardiac output and respiratory minute volume return to their preoperative values about fourteen days after operation. The blood oxygen content remains below the normal for about thirty days, while the minute blood volume for the right lung remains permanently elevated.

The left lung becomes completely atelectatic within at least three days after operation. Within four months there is definite and marked fibrosis of the collapsed lung. Its circulation gradually diminishes, being only 8 per cent. of the total cardiac output thirty-one days after collapse has occurred.

The right lung undergoes changes apparently similar to those following pneumectomy, i. e., a dilatation of the alveoli without any apparent increase in their number, without rupture of their walls and without fibrosis.

Protocol of Experimental Data

Dog	Time after Ligation	Carbon Dioxid Content		Oxygen Content		Minute Respiratory Volume	Percentage Carbon Dioxid in Expired Air	Total Minute Carbon Dioxid Output	Minute Blood Flow through Lungs	Pulse Rate	Respiratory Rate	Oxygen Capacity	Red Blood Cell Count
		Arterial	Venous	Arterial	Venous								
A1	Before	38.5	44	18.5	12.5	2,750	3.10	85.5	1,900	100	21	..	.....
	½ hour	32.5	36.5	12.5	6.5	3,100	2.25	70	1,750*	140	36	..	.....
	1 day	44.5	47.5	16.5	11	4,000	1.72	68.8	2,220*	120	40	..	.....
	7 days	41	45	18	11	2,800	2.72	76.1	1,920*	112	24	..	.....
	14 days	39.5	44	19	13	3,050	2.69	82	1,830*	120	20	..	.....
	21 days	42 5	46	16	9.5	2,400	2.56	61.5	1,750*	120	18	..	.....
	31 days	35	38	16	12	1,800	3.04	54.1	1,830*	100	16	..	.....
	48 days	39	44	22	16	2,650	3.43	90	1,820*	100	16	..	.....
A2	Before	41.5	45	18.5	12	3,300	2.75	90.6	2,590	120	24	..	.....
	½ hour	28.5	32.5	14.5	8.5	3,300	1.78	58.6	1,465*	140	36	..	.....
	1 day	35.5	39	14.5	8	4,600	1.37	63.2	1,800*	136	42	..	.....
	7 days	35	40	13.5	8	3,900	2.35	92	1,840*	120	24	..	.....
	14 days	34.5	39	12	7	3,700	2.38	88	1,950*	120	24	..	.....
	21 days	32.5	38	13.5	5.5	2,500	3.28	107	1,900*	130	24	..	.....
	28 days	34	38.5	12	7	3,100	2.08	64.5	1,430*	110	20	..	.....
	38 days	36.5	40	13.5	9	2,500	2.30	57.6	1,640*	90	18	..	.....
	50 days	35	39	14	8	2,450	2.25	55	1,580*	94	20	..	.....
A3	Before	38	42	20	14.5	2,600	3.17	80	2,000	108	16	..	.....
	½ hour	33	37	....	....	2,750	2.54	69.8	1,745*	108	18	..	.....
	1 day	48	42	22	16	3,000	2.18	63.6	1,590*	94	18	..	.....
A4	Before	41	44	18	12.5	2,950	3.74	110	2,750	130	20	..	.....
	½ hour	33	37	12	6	3,500	1.90	66	1,650*	170	24	..	.....
	1 day	40	44	12	5	2,100	3.28	69	1,725*	160	20	..	.....
A5	Before	32.5	36	19	14	3,100	2.29	71	2,020	120	24	21	5,500,000
	½ hour	30	34	15	10	6 000	1.67	100	2,500*	160	34	..	.....
	1 day	41	45	11	5	4,200	1.91	80	2,000*	140	28	24	7,150,000
	7 days	31	36.5	15	8	6,800	1.71	116	2,100*	144	28	23	6,720 000
A6	Before	36	40	22	16	8,700†	1.07	93	2,325	140	30	..	.....
	½ hour	40	44	16	....	8 800†	0.96	84.7	2,120*	160	28	..	.....
A7	Before	30	34	22	16	5,200†	2.35	122	3,050	120	30	..	.....
	½ hour	30	33	16	10.5	5,400†	1.40	75	2,500*	160	50	..	.....
P. A. Comp.		28	31.5	19	13	5,400†	1.88	102	2,910*	120	44	..	.....
A8	Before	38	43	18	12	5,100†	1.94	99	1.980	140	26	..	.....
	½ hour	37	41	10	4	5.100†	1.16	59.2	1,480*	110	28	..	.....
P. A. Comp.		38	42	17	11	5,250†	1.43	74.6	1,850*	100	28	..	.....
A9	Before	40	44	20	15	5,100†	1.79	91.7	2.540	130	24	..	.....
	½ hour	42	46	14	9.5	5,400†	1.06	57.3	1.430*	150	30	..	.....
P. A. Comp.		41.5	45 5	20	14.5	5,400	1.60	86.8	2,175*	120	20	..	.....

* Right lung after operation.
† By insufflation.

DISCUSSION

Dr. George J. Heuer, Cincinnati: The methods of study used by Dr. Andrus seem to open up another avenue of approach to the difficult subject of pneumothorax. He has, as nearly as is possible in dogs, reproduced the simplest condition, that of unilateral closed pneumothorax. He has found that in this condition the total lung volume is decreased 42 per cent.; but, in addition to this deleterious effect, the collapsed lung continues for a time to transmit 30 per cent. of the calculated cardiac output, blood which is surcharged with carbon dioxid. As a result there is a reduction of 30 per cent. in the carbon dioxid output of the expired air, an increase in the carbon dioxid content of the blood and a decrease in the oxygen content of the arterial blood. These effects are in part immediately compensated for as has been described. The observations made are of the greatest interest and importance and should be repeated, so far as is possible, on the human being. They give us for the first time, I think, an idea of what happens when pneumothorax is induced, both on the part of the individual who is the subject of the procedure and on the part of the lung which is collapsed. While it has been known, of course, that the collapsed lung becomes fibrotic and its blood supply diminished,

the observation that it at first transmits 30 per cent. of the cardiac output and, thirty days later, only 8 per cent., is, I believe, new and perhaps suggests one explanation for the tissue changes which take place. It suggests, also, the possibility of studying the changes which take place with the reexpansion of a long collapsed lung.

In addition to its interest as a contribution to the subject of closed pneumothorax, Dr. Andrus' paper forms the beginning of the study along similar lines of the more complicated subject of open or surgical pneumothorax. By the use of similar methods, it should be quite possible to study the effects of this condition on the lung in the open thorax, on the opposite lung, on the respiratory act and on the heart and great vessels, and, finally, on the organism as a whole. It should be possible to discover the limit of disturbances beyond which the organism cannot compensate. It should be possible to explain why different persons react differently to open pneumothorax, one being greatly upset, another unaffected. It should be possible to state not only that we need or do not always need pressure anesthesia in operations on the thorax but also precisely why we need or do not need it.

Dr. Leo Eloesser, San Francisco: I should like to ask Dr. Andrus whether, in his experiments, a pneumothorax remained after closing the chest or whether he inflated the lung so that there was no air left in the pleura. The question is raised whether the collapsed lung is anemic or hyperanemic. This has long been a subject of controversy. Whether one or the other is true seems to depend on the degree of collapse. Hyperemia probably follows a moderate collapse; anemia, a more complete one. I should like to ask whether the right or the left lung was collapsed. Was it not the left one? Yes. What is the normal relation between the blood supply of the right and the left lung?

Dr. Andrus: The right lung in dogs is slightly larger than the left.

Dr. Eloesser: I wonder whether the right and left lung each received 50 per cent. of the blood supply or whether the left lung, being smaller, naturally received a little less than the right one.

Dr. Edward W. Archibald, Montreal: The conditions brought about in the dog's chest by Dr. Andrus are similar to clinical conditions occasionally observed by surgeons. Dr. Scrimger of Montreal has been engaged during the past two years on the problem of massive lung collapse, and in particular with regard to its etiology. He opened the chest in dogs and ligated the bronchus of one lobe. He found that there resulted a collapse of that particular lobe. In the course of twenty-four hours, practically all the air contained in the lobe tied off had been absorbed, and the lung was in a state of massive collapse.

I merely mention these experiments as a possible small contribution to this problem, and with the idea that perhaps Dr. Scrimger and Dr. Andrus might do well to correspond. The work of both these gentlemen brings to my mind one question. Very frequently, in fact, I believe, always, one finds in the human being as one of the clinical signs of massive collapse, the expectoration of a thin watery sputum in large amounts, which I have interpreted as an edema of the lung; and I have asked myself whether that edema came from the collapsed lung or from the rest of the normal lung, which immediately tends to become, of course, emphysematous, as in animals. The retention of carbon dioxid in the blood, as mentioned by Dr. Andrus, has its correlative in the human being in the regular sign of cyanosis.

I thought possibly the correlation of these two things, in Dr. Andrus' experimental work and in clinical observations, might be of interest to the members.

DR. J. J. SINGER, St. Louis: The purpose of all experiments, of course, is the relation to the clinical manifestations and the use of the results in clinical treatment. The tying of the bronchus and the artificial collapse are not altogether analogous. As I understand it, it is almost impossible to produce a total collapse of the lung by artificial pneumothorax, and consequently one would expect differences in results. It has been known, for a long time, that a lung can be collapsed by artificial pneumothorax for five or six years and that the lung will come back to its original position after air is absorbed. It is a moot question now as to how long pneumothorax should be kept up, because it is stated by some that fibrosis occurs and the usefulness of the lung is impaired forever if collapse is too long.

I have seen patients that have been under pneumothorax treatment for eight years, and it is a very simple experiment to use a pneumothorax machine and draw off the air and see how the lung will, as I said before, retain its normal contour.

On the question of the return of circulation, I was not quite clear in my mind on the results that Dr. Andrus obtained, or the conclusion that 8 per cent. of the blood volume remained at the end of twenty-four days, and that, as the result of that reduced volume of blood, fibrosis occurs, in view of the fact that we see patients, say many years after, with a normal functioning lung when the pneumothorax has been discontinued. I feel that possibly the difference in the collapse in his experiment and in the human type of pneumothorax may account for that difference. I should like to have Dr. Andrus discuss that point.

DR. CARL EGGERS, New York: I am unable to add anything of an experimental nature to Dr. Andrus' paper, but I can say something about clinical chronic empyema which has a bearing on it. I have had an opportunity to see many patients and to operate on them, and have found that there is a marked difference in the ability with which lungs expand after decortication.

In the majority of acute empyema patients, the pus gradually develops, adhesions form, and, as the fluid increases, the lung becomes compressed by this fluid in a negative space. In some empyema cases on the other hand, in those in which the chest is opened before adhesions have formed, or in which perforation of a lung abscess produces a pyopneumothorax, the lung collapses. Judging from clinical observations, there is a very decided difference between a compressed and a collapsed lung. In the first group, the circulation is never seriously interfered with, and as soon as a decortication is done, the lung will expand, even after years of compression. In the second group, on the other hand, there is serious interference with circulation, fibrosis gradually develops, and, if such a lung is allowed to remain in that collapsed condition long, it will not be able to reexpand, even after thorough decortication. It is this group of cases in which a collapsing operation of the chest wall is necessary to obliterate the cavity.

DR. WILLIS S. LEMON, Rochester, Minn.: It seem to me that Dr. Archibald's reference to collapse of the lung is a pertinent one. This point has been fully described by Pasteur, Bradford, Scrimger, Bristol and others, and their description pictures a condition similar to that described by Dr. Andrus. Clinically, one cannot diagnose a massive collapse of the lung unless the diaphragm is raised on the collapsed side, the chest on that side flattened, and the heart drawn to the affected side. If this experimental work produced other than a massive collapse, then one would expect a different complex, namely, that the diaphragm, if anything, would be lower, the chest wall would not be particularly compressed, and the heart would be in the opposite side.

Dr. William D. Andrus, Cincinnati: In regard to the relative amount of blood flowing through the two lungs, we have simply assumed from the relative size of the lungs and of their vessels that each lung was transmitting about 50 per cent. of the blood; it seemed to us a fair conclusion.

I would say in answering Dr. Archibald's question regarding the edema after the collapse that in only three animals did we do an immediate necropsy and in those animals another procedure had been carried out. While we did see edema in those three cases, we cannot, I think, be sure that it was the collapse which had produced it. In these animals, when we ligated the bronchus, we placed a compression clamp, such as that described by Drinker and Peabody, around the pulmonary artery on the same side. This was left loose about the artery, and the shaft of the clamp brought out the lower angle of the wound. Then, after we had made our first series of determinations following the ligation of the bronchus, we compressed the artery with the clamp and shunted all the blood through the aerated lung. This procedure produced some very interesting effects, as we have mentioned in our paper.

Regarding the degree of pulmonary compression obtained in these experiments, I may say that we have tried to produce the same degree of compression that would have been produced had the dog's chest suddenly been widely opened on one side. The lung collapses but slowly following bronchial ligation ordinarily, since the contained air is 80 per cent. nitrogen, which is taken up very slowly as the blood is already saturated with it. However, if one simply insufflates with oxygen for a few moments before ligating the bronchus, complete atelectasis can be produced in a very short time.

Regarding the fibrosis which we found in these experiments, I think we cannot say whether this is due to reduced blood supply or vice versa. These experiments constitute what we hope may be the first link in a chain of experimental work on the fundamental abnormal physiology of pneumothorax. What we are trying to do is to analyze this problem into its component parts, study the parts separately, and then piece together the evidence obtained in the hope that we may throw further light on the subject.

THE REACTION OF THE PLEURA TO PARTICULATE MATTER, ESPECIALLY IN THE PRESENCE OF PNEUMOTHORAX

A PRELIMINARY REPORT *

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CHICAGO

About a month ago, I presented, at a meeting of the Chicago Surgical Society, some preliminary reports of expermiental work I had been carrying out on the absorptive power of the pleura. Dr. Hedblom, who was present, thought that these notes might be of sufficient interest to read to you.

While carrying on some experiments which had nothing to do with this present problem, I injected 1 c.c. of India ink into the pleural cavity of a number of dogs. When these dogs were necropsied some four days later, I was surprised to see that the pleural cavities were clear, and that, except for the black lines marking the intercostal lymph channels and the staining of the lymph channels on the diaphragm and the pigmentation of the mediastinal lymph nodes, no trace of the India ink was to be found.

I repeated these experiments on rabbits and here again found that, if I injected 1 c.c. of India ink into a normal pleural space, four or five days later, both visceral and parietal pleurae were unstained, that the intracostal and superdiaphragmatic lymph channels were deeply pigmented, and that the mediastinal glands and the hilium glands had absorbed large quantities of the India ink. The microscopic section revealed the fact that the pleurae were normal, and that, except in one place where a microscopic deposit of India ink was found in the visceral pleura and from which small India ink particles were being apparently carried into the lung tissue, no ink particles were to be found in the lungs. In other words, it seemed clear that the normal pleura was able to absorb 1 c.c. of India ink without producing any apparent reaction.

I then took a second series of rabbits in which, besides injecting the 1 c.c. of India ink into the right pleural space, I injected 20 c.c. of air. In some I refilled the pleural space with air two or three days later. On necropsy now, a striking difference presented. The pleural space,

* From the Nelson Morris Memorial Institute for Medical Research of the Michael Reese Hospital.

instead of being clear, was stained, the stain being held in a thin film which covered both the visceral and parietal pleurae. This film in some areas was easily stripped, in others was adherent. The lymph channels and the lymph nodes were again injected but apparently to a lesser degree than in the first series. The lungs on cross section were found to be free of stain. Microscopic sections showed that the pleurae were thickened, that many new fibroblasts were present and that in some areas new blood vessel tufts had been formed. Within this tissue were

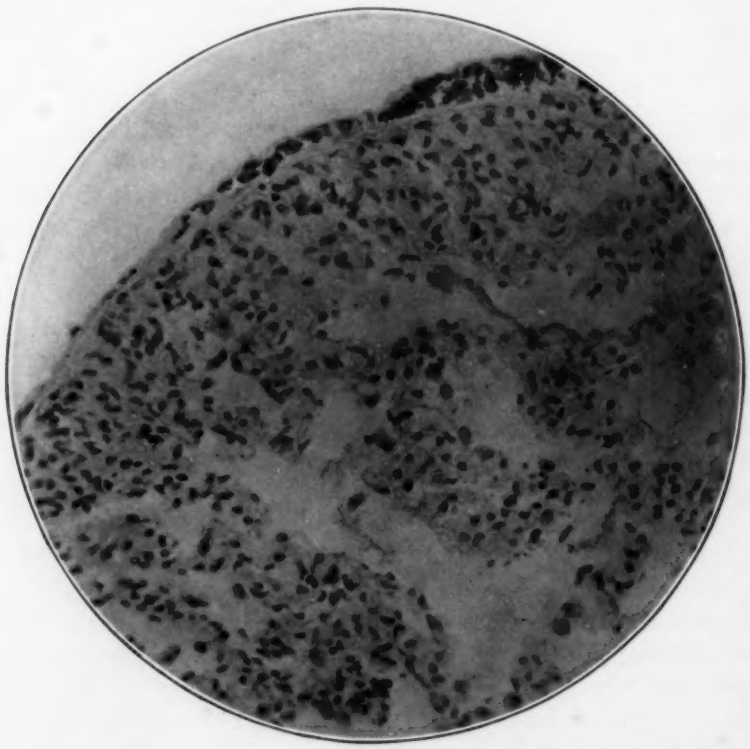

Fig. 1.—Section of the pleura five days after 1 c.c. of India ink had been injected into the pleural space, but no pneumothorax produced. The thin, normal, unstained pleura should be noted. In the upper angle can be seen one of the rare deposits of ink granules.

found large deposits of India ink. It was of interest to note that in spite of the fact that these large numbers of granules were held captive in the pleura, no more and perhaps fewer India ink granules had been carried into the lung tissue by phagocytes.

From these two sets of experiments, it seemed apparent that the presence of a pneumothorax had so altered the physiology of the pleural space that the pleura was no longer able to dispose of the 1 c.c. of India

ink. It seemed to me that a plausible explanation for the inability of the pleura to absorb the India ink might be the proliferative reaction which was found. These experiments seemed to explain many well known experimental and clinical facts. Notzel, years ago, showed that the normal pleura was very resistant to bacteria, but that in the presence of a pneumothorax much of this resistant power was lost. Furthermore, clinically, we realize that, in the presence of a pneumothorax, infections of the pleura are much more stubborn. Numerous authors

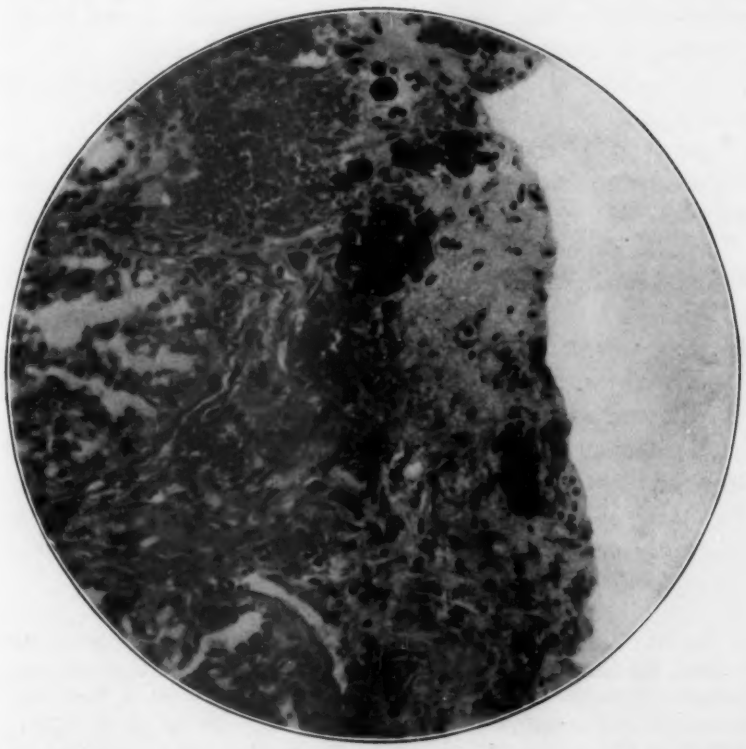

Fig. 2.—Section of the pleura five days after 1 c.c. of India ink had been injected and a pneumothorax produced. The thickened pleura and the marked reaction should be noted. Young fibroblasts and newly forming blood vessel tufts can be seen; there is a large accumulation or deposit of India ink granules.

have pointed to the dangers of reinfection after an empyema in unobliterated cavities. For these reasons, I had the temerity to present my preliminary reports.

In the last few months, I have carried on several experiments in an attempt to find out just what produced this proliferative reaction of the pleura. The presence of air alone in the pleura did not account for my findings. In a series of rabbits in which I produced a pneumothorax

by injecting air into the pleural cavity with a syringe, I kept the pneumothorax for two weeks by means of frequent refills. I found at the end of this time that there was very little change in the pleura. In many of the microscopic sections of the visceral pleura, there would seem to be a slight thickening, but this thickening was evidently due to the atelectatic condition of the underlying lung and not to any proliferative reaction of the pleura itself. No new fibroblasts were seen, nor newly formed blood vessels.

In another series, I injected sterile physiologic sodium chlorid solution to see whether the mere fact of keeping the pleural layers apart without the irritant effect of the air would produce a change. I injected as much warm, sterile physiologic sodium chlorid solution as the rabbits were able to stand into the right pleural space, repeating my injections every day, and, after a week, found that except at site of the needle punctures, no proliferative reaction had been produced in the pleura. I then repeated these experiments but injected 1 c.c. of India ink with the sterile saline solution on my first injection, and at the end of a week found conditions very similar to those where I had injected air and India ink.

In other words, it seems to me from these few preliminary studies that neither the mere presence of air, nor the separation of the pleural layers will in itself produce changes in the pleura; but that, on the addition of some particular matter, such as India ink; the pleura is found to be no longer capable of resistance as it normally was, but that the resistance of the pleura to particulate matter is so lowered that it can no longer absorb.

DISCUSSION

DR. FRANK C. MANN, Rochester, Minn.: Dr. Bettman has really left nothing to discuss. He has stated some very interesting and important experimental results. I wish to recall two things in this regard. It has been so often emphasized in this society that thoracic surgery is more or less a child of the war in which the surgeon has overcome the psychologic feeling that he had in regard to the pleural cavity and now enters it with impunity. However, some of the very first experimental work that was done in intrathoracic surgery was done by Carrel, and, most of his papers give the details of how carefully he went into the pleural cavity.

I remember the first experimental work we did in which we entered the thorax; we attempted to follow the Carrel technic. He used oil silk which was a very good grade of Japanese silk, very carefully sterilized in oil, and he used extreme care in entering the thoracic cavity to take care of the pleura.

I think that Dr. Bettman's paper is timely in recalling that the pleura is a very delicate epithelial cell lined cavity and that we must use the greatest care in treating it, because these cells are easily injured and they have a very definite function in lining this cavity and in taking care of the proper absorption from it as well as keeping it oiled, so to speak, by the solution which it secretes.

My own work which might have a little bearing on Dr. Bettman's paper has been in regard to the irritation produced by just such little things as a piece of cotton or a piece of gauze.

Dr. Duff S. Allen, St. Louis: I think that Dr. Bettman's paper is a step in the right direction. The reaction of the pleura to different substances has been studied to some extent, but not completely. Substances which form a perfect solution, such as electrolytes and sodium chlorid, are absorbed by the pleura in a different way than are the solids. India ink is a suspension of fine particles of carbon, and there is, therefore, a suspension of solid bodies. They are absorbed by phagocytic action, as Dr. Bettman states, and certain things will delay the absorption. The same is true of the peritoneum. Metchnikoff, I believe, was the first to study this action in the peritoneum when he placed milk on the diaphragm, and he was able to demonstrate to his satisfaction that there were stoma through which the particles of milk were passed.

Storna was first to work with electrolyte, or one of the first. Dr. Beck was the first to try solid particles in the form of blood corpuscles in the peritoneum. Now, there are certain things that will delay this action not only in the peritoneum but also in the pleura. Those things are mainly inflammatory reactions, such as acute inflammation that is due to bacteria or chronic inflammatory processes; and also, in the peritoneum, the slowing of the peristalsis will delay the absorption action.

I wonder if Dr. Bettman has tried electrolytes. If the electrolytes are hypotonic, then the absorption of the electrolyte does not begin until enough water has been drawn out to make it isotonic. The absorption of this, instead of being phagocytic is by osmosis or by dialysis.

That the pleura exposed to the air over a long period of time is not always rendered impermeable to electrolytes I think is well proved by a patient, in the Barnes' Hospital at the present time, who has had an opening in the thorax for seven months. He has a tuberculous empyema which was drained by a resection of the rib. Smears showed many tubercle bacilli in the pus. We irrigated him with mercurochrome. Three days later, the urine was stained with mercurochrome, and even the sweat was pink. There is an absorptive power in the pleura, although undoubtedly, in his weakness and in view of Dr. Bettman's finding, one can see a reason for the delay; still we know that there is an absorptive power left in this pleura.

Let me say again that I think that this paper and this work are along the right line. Future work may throw further light on this important subject.

Dr. Carl Eggers, New York: The importance of Dr. Bettman's observation is to be found in its relation to clinical cases. Does it perhaps throw light on certain unexplained temperatures occasionally observed in acute empyema?

If one drains an ordinary empyema, which has run its acute course, and in which thick pus has formed, the temperature usually comes down promptly and remains down, and the patient at once feels relieved. There is no respiratory difficulty.

If, on the other hand, one drains an acute empyema with an extensive exudate which is thin in character, the kind usually found in the early streptococcus cases, there is considerable respiratory difficulty, with more or less collapse of the lung, and the course is not such a smooth one. Even with closed drainage, the difficulty is not always entirely overcome, and one at times does get a moderate pneumothorax with partial collapse of the lung. It is in these patients with a pneumothorax in whom we have repeatedly observed unexplained temperatures that sometimes run a course of weeks.

Careful physical examination has given no explanation for it; there have been no complications that could be determined; the roentgen-ray examination has not helped us at all; there has been no intrapulmonary focus; the drainage opening has been at the dependent part of the cavity, and the latter has been kept clean with the aid of chemical solutions.

I have often been asked how to explain these temperatures, and have been rather at a loss to do so in the absence of any positive evidence. My answer has always been that it was probably due to the presence of streptococci in the tissues, for the late Dr. Dunham of the empyema commission showed very clearly that streptococci in empyema travel not alone by the blood stream but also that they have the power to travel along the tissue spaces and may so invade the opposite pleura, the pericardium or the peritoneum.

Now, Dr. Bettman's experiments show that there is stagnation in the lymphatics of the visceral pleura in cases with a pneumothorax. In his experiments, it was a dye which blocked the vessels, and it is not difficult to believe that, in clinical cases, it is the bacteria which choke the lymphatics in the same way. This stagnation is due to disturbed circulation, and that in turn to disturbed respiration. While thus held back, the organisms may rapidly increase in number and be responsible for prolonged temperatures.

Applied to practical medicine, Dr. Bettman's observation is further evidence of the importance of avoiding an acute pneumothorax. It may therefore be timely to emphasize once more the teaching not to operate in streptococcus cases until thick pus and adhesions have formed and the pneumonia has subsided, except in urgent cases, and in those to use one of the closed methods to prevent collapse of the lung.

Dr. Willis S. Lemon, Rochester, Minn.: I should like to put my discussion in the form of a question: Is the condition entirely chemical or is it mechanical? The lung cannot be reduced to a fraction of its normal size without likewise reducing the size of the vessels that carry away the materials from the pleural cavity. Any foreign material, such as India ink, is probably carried away by phagocytic action, and consequently reduction in the size of the lymphatics may be sufficient to prevent this conveyance of particulate matter and explain, therefore, the accumulation within the pleura itself. Such an accumulation formed in the pleura should provide sufficient irritation to thicken it, and masses of particulate matter should be held encased. This explanation might be offered as well as the chemical explanations given by Dr. Bettman.

Dr. Willy Meyer, New York: Dr. Bettman's investigations are not only interesting, but of clinical importance. His experiments are particularly convincing because he used the India ink. When he injected the ink alone, nothing occurred; when he added a pneumothorax, then the observation explained by him set in.

In a rather reverse fashion clinicians have already proved the correctness of his findings in seeing the effect of the occurrence of a pneumothorax, for instance, in the after-treatment of acute empyema, also the difficulty of curing a spontaneously occurring pneumothorax in the tuberculous.

We have done, at our hospital, in a number of instances, the so-called closed treatment of acute empyema by pushing the trocar into the cavity and then introducing through the cannula the drainage tube, in air-tight fashion. Everything went perfectly splendid in some of these cases, without any rise of temperature, as long as this air-tight drainage was left undisturbed. When siphonage was discarded and open drainage substituted, irregular, often high, fever set in, the cause of which we were unable to explain. Slowly the patients

recovered. Dr. Bettman's investigations throw some light on the occurrence of these prolonged elevations of temperature.

In some of the cases of a tuberculous spontaneous pneumothorax, the formation of the exudate is scant. To cure the pneumothorax, the closure of the pulmonary fistula is a matter of necessity. This is often a difficult task. Knowing from experience that conditions for the closure of such a tuberculous lung fistula are better, if a large exudate has developed with the pneumothorax, because the exudate compresses the lung, thickens the pleura and induces absorption of the air in the pneumothorax cavity, Spengler of Davos proposed, almost twenty years ago, to produce a larger exudate artificially by injecting a few cubic centimeters of a one-half per cent. solution of nitrate of silver into the pleural cavity. Later on, he advised injecting 30 c.c. of a sterile glucose solution in order to reduce the subsequent reaction. After a short rise of temperature, he saw the exudate become reduced and at last totally absorbed; the pulmonary fistula closed.

Dr. Leo Eloesser, San Francisco: Experiments on the production of lymphatic obstruction by scar have shown that an aseptic scar will produce a partial stenosis of the lymphatics such as Dr. Bettman has found in the pleura; infectious inflammatory processes seem to cause a complete block.

Dr. Emil G. Beck, Chicago: I wonder what effect the artificial pneumothorax, such as is carried out in tuberculosis, would bear on the experiments of Dr. Bettman.

The theory which was advanced by Dr. Eggers is a very sound one and I have been able to convince myself of the correctness of his theory many times.

We have seen a great many cases of chronic empyema which were of a tuberculous origin. While we could not demonstrate tubercle bacilli in the secretions by microscopic examination, we were surprised that, when these pleural cavities were injected with a 33 per cent. bismuth mixture, we could, after two or three hours, demonstrate tubercle bacilli in very large numbers. We have verified this fact in about ten cases, which I published at that time (Diagnose, chirurgische Behandlung und Verhutung von Fistelgangen und Abseesshohlen, Beitr. y. Klin. Chir. **62**, Part II, 1909). To explain this phenomena, we thought of this possibility, that through the introduction of an irritating substance, such as bismuth, we could produce a reaction in the thickened pleura, a leukocytosis, and that by this leukocytic action tubercle bacilli were thrown out. These bacilli differed, however, from the usual. They looked like a chain of three or four streptococci. That fact proves to us that when an irritating substance was put into the thick pleura, which held micro-organisms locked in the connective tissue, the thickened pleura might be thrown out by such substance as India ink.

Dr. Ralph B. Bettman, Chicago: As I said, I undertook this work to check up on some of the findings that I have noted in acute empyema. I have been a very ardent adherer to the closed method although I have had the same results occasionally that Dr. Willy Meyer and Dr. Eggers have mentioned, namely, that after the apparently normal convalescence, there would be a reinfection. In looking for an explanation, I happened on these findings. The significant thing, in my experiments, seemed to be that in a normal pleura there was no reaction with India ink, while, when pneumothorax had been introduced, the India ink was kept in the pleura and there was a marked reaction. The explanation for it, as I said, I am not at present willing to give. There seems to be a definite lymphatic block during the proliferation of the pleura. That it may be due to mechanical factor, as

Dr. Lemon states, I am very well aware. If one does produce a pneumothorax, one is not only causing a lymphatic block which blocks up — I don't like to say stoma because we doubt whether there are regular openings to the lymphatic channels — but at least blocks up the lymphatic channels and also reduces the space through which the lymphatics can absorb material.

It is of interest to note that I have tried, as Dr. Allen suggested, colloidal materials, and I have found either no, or very little, reaction. The India ink granules seemed to me to fit the experiment very much better than a colloidal material because they are more similar to bacteria; they are definite suspensions, and I thought the reaction would be more typical. The injection of bacteria instead of India ink confused the picture on account of the great septic inflammatory reaction.

CERVICAL SYMPATHECTOMY IN ANGINA PECTORIS

A REPORT OF THREE CASES

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Angina pectoris is not the name of a disease. It refers to certain subjective phenomena which occur probably in a number of morbid conditions of the heart, its intrinsic arterial system and the aorta. The clinical picture may be of two distinct types, the first accompanied by attacks of substernal spasmodic choking pain, many times described as "pressing" and often running down the arm, usually the left; and, second, a type in which the localization of the pain and distress is in the upper part of the abdomen. Here, too, the pain often shoots down one or the other or even both arms. It is frequently accompanied by belching of great quantities of gas. As a rule, an attack partakes of some of the symptoms of both types, but one or the other predominates. There may be all grades of severity, the worst being accompanied by a most alarming sensation of impending death. In any attack of angina pectoris there is danger of death. It is not necessary here to go into a minute description of the purely medical aspects of the syndrome. For this there are the standard books on medicine. It is worth while, however, to call attention to the fact that there may or may not be vascular hypertension. In the cases that I here report, all three of the patients suffered from high blood pressure. It also should be remembered that seizures of angina are often mistaken for what has been called acute indigestion, which may include almost any abdominal condition accompanied by cramps, the commonest of which is biliary colic,[1] and that the the reverse is also true, abdominal colic being occasionally mistaken for an attack of angina. It must be understood, too, that true angina may be present in cases of acute abdominal disease accompanied by colic.

The conditions producing the angina are most often encountered in men over 45 years of age, and the attacks are apt to follow muscular exertion and to appear after a heavy meal. Exercise following the ingestion of food is almost sure to bring on an attack. There is myocarditis and often coronary arteritis with or without thrombosis. From the surgical standpoint, the important thing is that in the fatal cases of angina pectoris it is rare to find complete coronary obliteration. While

1. Willius, F. A.: Ann. Surg. **79**:524 (April) 1924.

spasm of these vessels cannot be proved, it would explain certain fatalities in which their lumina are apparently not sufficiently occluded to cause asphyxia of that part of the muscle included in their distribution.

Clifford Allbutt [2] is of the opinion that disease of the aorta is present in about 90 per cent. of the cases, and that the anginoid phenomena have their origin in the outer vascular covering in which are the sensory end organs that regulate blood pressure. He believes that sudden death is often caused by inhibition of the vagus by the shock of the pain.

It seems reasonable that any procedure which would tend to prevent arterial spasm in these important vessels would also prevent the accompanying painful sensations. Delorme, in discussing a case in which Tuffier had wrapped an aortic aneurysm with a strip of fascia lata, believed that the subsequent relief was probably due to the freeing or removal of the sympathetic branches which was necessary in exposing the aneurysm, and he even suggested that vascular sympathectomy would be worth performing for the relief of the pain of aortitis.

In view of the success that has been achieved by the comparatively simple procedure of cervical sympathectomy, it is surprising to learn that such severe operations as posterior rhizotomy of the second, third and fourth thoracic nerves has been recently suggested for the relief of angina pectoris. Goodall and Rogers had revived this idea first conceived by Goodall and Earle [4] three years before Jonnesco's work on the sympathetic in relation to this disorder. The latest suggestion of Goodall and Rogers [3] is nerve blocking with alcohol or procain, but they do not state that this could be accomplished without laminectomy. They refer also to recent papers by Danielopolu of Bucharest on the section of the left segmental nerves or their injection with procain to relieve the pain of angina.

Jonnesco, in 1916, attacked the sympathetic system from its cervical side and performed his operation of cervical sympathectomy on a patient removing all the ganglions of one side together with the first thoracic. The patient was followed for four years, and his heart action and respiration remained apparently normal. With increasing experience, Jonnesco became enthusiastic and operated many times. In 1923, Coffey and Brown [5] reported six cases in which cervical sympathectomy had been performed by them, with one death and great improvement in

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2. Allbutt, Clifford: Lancet **1**:883-938 (May 5) 1923.

3. Goodall, J. S., and Rogers, Lambert: Some Surgical Problems of Cardiology, Technic of Mitralotomy, Am. J. Surg. **38**:108 (May) 1924.

4. Goodall, J. S., and Earle, H. G.: Arch. Middlesex Hosp., 1913; Earle, H. G.: quoted by Goodall and Rogers (Footnote 3).

5. Coffey, W. B., and Brown, P. K.: Surgical Treatment of Angina Pectoris, Arch. Int. Med. **31**:200 (Feb.) 1923.

the other five. In some of their cases, there was complete relief on extirpation of the superior ganglion alone, and this was not accompanied by Horner's syndrome, although it was stated that there was contraction of the corresponding pupil.

A striking case has been reported by Bacon,[6] who operated successfully on a man of 78 who had suffered three years from anginal attacks in the severest form. After sympathectomy, this patient was able to walk 3 miles at a time without resting and without discomfort.

A report of my own cases follows:

CASE 1.—From the experience of Jonnesco and that of Coffey and Brown, it appeared to me that the operation in cases properly diagnosed was eminently justifiable, and when a case presented itself to me in the person of one of my college classmates I did not hesitate to place the matter before him together with my impressions. I referred him to Dr. S. A. Levine of the Peter Bent Brigham Hospital in order to make sure of the diagnosis and the prognosis. The fact that my friend's father had died of the disease added considerably to his nervousness. Dr. Levine reported that the patient's weight was 172; his blood pressure 190 systolic, 106 diastolic; that the action of the definitely enlarged heart was regular, the first sound being slightly diminished in quality; that there was a slightly diminished vital capacity, and that the Wassermann reaction was negative. The electrocardiogram was that of a case of hypertension. The first of the attacks was on Dec. 31, 1923, and they became very frequent, occurring almost daily, and of an alarming though not of the severest type. Dr. Levine believed that the possibility of prompt relief would be greater with operation than with purely medical treatment. The patient came to New York where I observed him for a few days. His attacks had become very frequent, as many as seven or eight in a day, but they were somewhat controlled by nitroglycerin in small doses.

Jan. 21, 1924, I operated in Mt. Sinai Hospital, beginning with local anesthesia, but being obliged to change to general nitrous oxid, oxygen and very little ether (Eliasberg), because of the patient's apprehension. The procedure was complicated by two anomalies: the first, not particularly important, was the absence of the external jugular vein; the second, most annoying, was the absence of the left internal jugular vein. I had not, at the time, heard of this vascular anomaly and wasted about fifteen minutes making perfectly sure that there was indeed no vestige of the vein. The sympathetic cord was picked up in the upper part of the neck just behind the internal carotid artery. It was identified by its middle ganglion, and was followed upward until it expanded into the characteristic form of the superior ganglion. This structure was raised with forceps and its branches were divided with scissors. The ganglion was then seized and slowly avulsed from its attachments. Using the ganglion as a handle, the nerve was dissected to the second ganglion, down, but not so far as the inferior ganglion. It was finally divided with scissors and removed. Although the third ganglion had not been seen, there was immediate contraction of the left pupil and ptosis was noted as soon as the patient

6. Bacon, J. H.: Left Superior Cervical Sympathectomy Under Local Anesthesia in Angina Pectoris, J. A. M. A. **81**:2112 (Dec. 22) 1923.

had aroused from the anesthetic. The wound was closed with metal clips and a forty-eight hour drainage established by a small tube at the lower angle of the wound.

The patient bore the operation well, and the pulse remained normal throughout. That night there were very slight, but almost continuous anginoid attacks which were relieved by nitroglycerin and finally by a little morphin. They ceased, and up to the present time (five months) there has been no angina. There was slight infection at the middle part of the wound, but the removal of two of the skin clips was followed by uninterrupted healing. The blood pressure just before operation was 160 systolic. Three days later, it had fallen to 105 systolic, 50 diastolic. It gradually rose in three more days to 130 systolic, 60 diastolic, with the patient sitting, and at present is 156½. He was discharged from the hospital two weeks after the operation, and was referred back to Dr. Levine for further medical treatment.

This patient had been very skeptical as to the final results, but in a letter, dated April 26, 1924, more than three months after the operation, he was most enthusiastic and said that he lives about as he did before he had the angina, eating and exercising in a normal manner. He has been told, however, that he must not overdo, and that he must remain under medical care. Horner's syndrome does not annoy him, and there is no sweating of the face. He states that his left arm does not feel normal, and that there are sore spots on the inner and outer ends of the left collar bone, including the top of the shoulder, and the upper part of the scapular blade. There are anomalous sensations in the tongue, the ear and the jaw on the left side. These, however, are not particularly annoying. An interesting observation made by the patient is that there is a painful sensation in the tongue on taking any acid or salty substance in his mouth. Improvement has been progressive to the present (November, 1924).

CASE 2.—Mrs. R. G., aged 49, who had been under the care of Dr. Louis Miller, came to see me, Feb. 11, 1924. The past history was irrelevant. For six years, she had been having anginal attacks, mostly nocturnal, at least once a week and sometimes as often as twice in a single night. The pain was often continuous for as long as two hours, and usually required hypodermic injections of morphin for relief. The sensations were in the left chest and arm. The case was one of hypertension, the systolic pressure often running higher than 300. Electrocardiographic examination by Dr. B. S. Oppenheimer indicated abnormalities associated with hypertension, that is, inversion of the main deflection in Lead III, indicating left ventricular predominance; rather high voltage of the main deflection in Leads I and III; simple tachycardia; the T waves in dissimilar directions to the main deflection in Leads I and III, i. e., T inverted in Lead I and upright in Lead III (Fig. 1). She entered Mt. Sinai Hospital, and at 5:15 p. m., February 23, she was seen in an attack in which there were evidences of severe precordial pain, nausea and retching with the bringing up of a little foamy mucus. The pulse rate at this time was 160; breathing was deep and noisy; the face was flushed and the forehead perspiring. The next day, the blood pressure was 262 systolic, 130 diastolic.

February 25, I operated, using ether by the open drop method. I feared nitrous oxid on account of its tendency to increase the vascular tension. Just before the anesthetic was given, the blood pressure was more than 260. The pulse rate was 100. The patient had received a hypodermic dose of morphin. In order to reduce the pressure during operation and eliminate part at least of the danger of cerebral hemorrhage, I segregated the blood of the lower

extremities by ligating the thighs close to the body. This brought down the reading to 240 systolic, a reduction less marked than is usual by this method. The ligatures were left on during the operation, and, immediately afterwards, the systolic pressure was 230 before the ligatures were removed. The operation was performed in the usual manner through an incision along the posterior border of the left sternocleidomastoid muscle. The neurovascular bundle, together with this muscle, was retracted, and the cervical sympathetic cord was easily found. It was encircled with a retracting ligature of catgut for convenience in handling, and was dissected free from its bed up to the

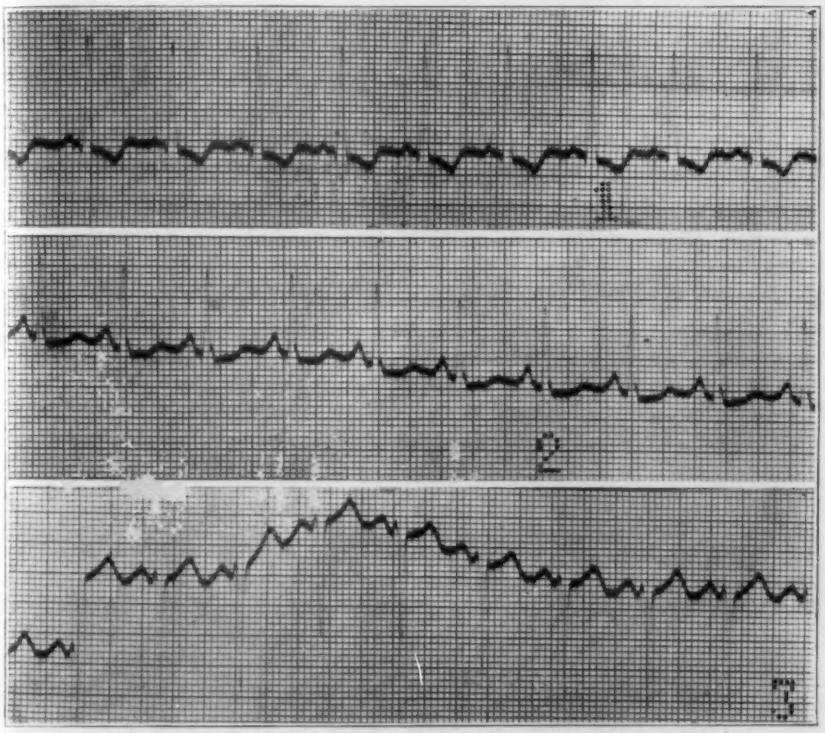

Fig. 1 (Case 2).—Preoperative electrocardiogram.

superior ganglion. This was loosened from its adhesions and avulsed. Using the ganglion now as a handle, I dissected the nerve down beyond the middle ganglion and divided it below this point, no attempt being made to remove the lowermost one. The pupils showed no asymmetry, and I thought that this was because the preliminary dose of morphin had contracted them, but both sides responded to the instillation of homatropin, and there never was any contraction of the pupil or ptosis. Very slight pupillary asymmetry was observed five or six weeks later.

Forty-eight hours after the operation, the systolic pressure had receded to 205. Six days after the operation, it had risen to 251. The pulse rate remained at about 100. Healing was by primary union, and there were no further attacks. The patient slept well all night, which she had not done

for six months before. I have suggested to her that, on account of the rising blood pressure and slight sensations of cardiac uneasiness, a similar operation on the other side would be advisable. The patient lives on the third floor of a "walk up" apartment, and ascends the stairs with little discomfort.

Case 3.—Mrs. A. K., a woman, aged 49, who also suffered from hypertension, for many years, had had anginal attacks. They became increasingly severe until every week there were from two to four, some of them lasting several hours. This was one of the cases in which abdominal disease was simulated. The patient belched enormous quantities of gas, complained of pain in the upper part of the abdomen and in the precordial region, the

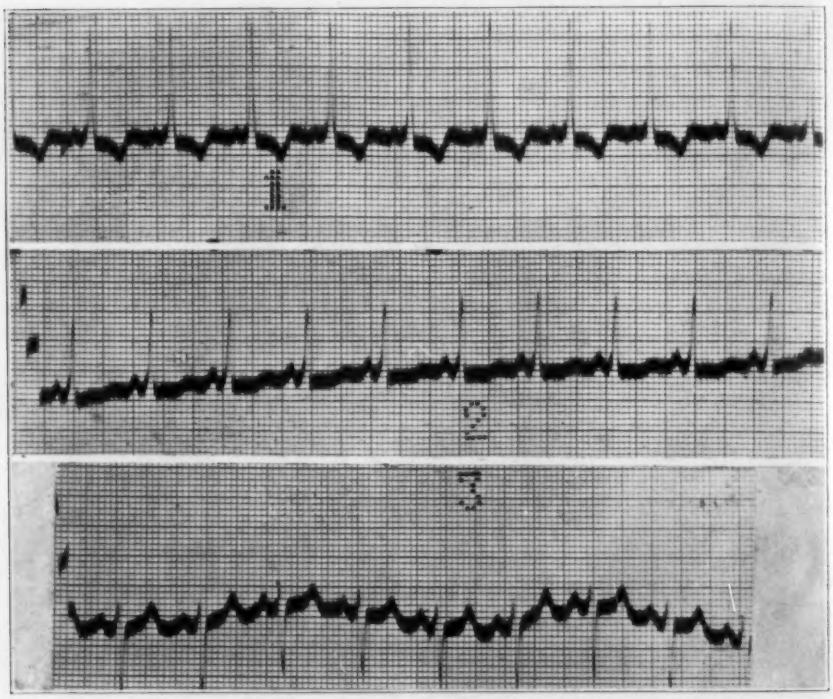

Fig. 2 (Case 3).—Preoperative electrocardiogram.

painful sensations passing down the left arm. After a particularly heavy series, her physician, Dr. Tripler, advised complete rest in bed, and she was treated in this way for two weeks before I saw her. Dr. Morris Manges had been called in consultation and had advised operation.

She entered Mt. Sinai Hospital, March 13, 1924, and was examined by Dr. B. S. Oppenheimer, who confirmed the diagnosis and made an electrocardiographic study. There was a high voltage in all leads. There was marked left ventricular preponderance. The T wave was angularly inverted in Lead I, and upright and peaked in Lead III, a type commonly seen in hypertension. In the course of examination, the cardiac pacemaker abruptly changed to a lower level, resulting in a nodal rhythm for a short period (Fig. 2). The blood pressure, on admission, was 195 systolic, 110 diastolic.

but, before admission to the hospital, it had run to more than 300 systolic. The pulse rate ranged between 84 and 100. Roentgen-ray examination of the heart indicated hypertrophy of the left ventricle with moderate dilatation. There was diffuse dilatation of the thoracic arch, probably due to atheroma. The apex of the right lung showed a number of scars apparently from healed tuberculosis. The urine contained a large trace of albumin, a number of pus cells, a few hyaline casts and about 1.5 per cent. of urea, the total daily quantity being about 40 ounces (1182 c.c.). A few months before, the patient had been operated on for left mastoid disease. There was a resulting healthy

Fig. 3 (Case 3).—Horner's syndrome on the left after left sided sympathectomy.

scar without sinuses. The day of her entrance into the hospital, she had a severe attack and, two days later, March 15, I operated under general anesthesia, using nitrous oxid and a little ether, administered by Dr. Branower. Just before she took the anesthetic, the blood pressure was observed, and it was higher than could be measured by the hospital apparatus which ran only to 260. The operation proceeded as in the other two cases, but it was found that the left sympathetic nerve had been retracted with the neurovascular bundle. It was, however, easily identified and followed up to its superior ganglion which was loosened, with the aid of dissecting scissors, and avulsed. The

nerve was then followed down beyond the middle cervical ganglion and there divided, completing the resection. I did not remove the lowermost ganglion because in the other two cases good results had followed without this additional procedure, and Coffey and Brown had succeeded by the removal of the superior ganglion only. The wound was closed with skin clips and a small drainage tube to get rid of the early discharges. Immediately after the operation, the blood pressure was reduced to 195 systolic; the next day it rose again to 260, and the patient had vague sensations of discomfort but no angina. The second night, however, there was a sharp attack, and it was clear that the opposite

Fig. 4 (Case 3).—Double Horner's syndrome after double sympathectomy; facial symmetry restored.

sympathetic would have to be resected. The patient requested that this be attempted with local anesthesia and accordingly, March 21, six days after the first resection, the left wound being nearly healed, the same procedure was carried out under local anesthesia on the right side. The nerve was then followed down to the lower ganglion, which was carefully isolated and cut away with scissors. During this part of the operation, the patient suffered pain in the chest and, although she was perfectly quiet during the entire time, she stated that the part of the procedure on the inferior ganglion was accompanied by severe pain.

The blood pressure after this operation varied from 205 systolic, 105 diastolic, to 235 systolic, 110 diastolic, and the pulse from 72 to 108. After the first operation, there was distinct Horner's syndrome, but no sweating of the face. The appearance is shown in Figure 3. After the second operation, the face became symmetrical because of the double Horner's syndrome (Fig. 4). Examination of the fundi by Dr. Schlivek showed the nerve head of the right eye negative. The arteries were narrow with a brilliant reflex, and caused indentation where they crossed the veins. To the temporal side of the nerve head, there were numerous minute spots of degeneration in and about the macula, and to its temporal side there were numerous hemorrhages, fresh and old, and spots of fibrous exudate. In the left eye, the nerve head and vessels were the same as those on the right, but the hemorrhage and spots of degeneration were very few.

With the exception of one slight attack a few days after the last operation, designated by the patient as "indigestion," but which I believe to have been anginoid, this patient has been free from cardiac symptoms. She expresses herself as extremely grateful for what she calls "complete relief."

It seems almost unnecessary to call the attention of surgeons to the great importance of the unequivocal diagnosis of angina pectoris in deciding to operate for its relief. I believe that the concurrence of an expert cardiologist should always be secured before attempting surgery of this kind. Whether there are contraindications or not toward operation in typical cases of angina pectoris cannot at present be stated. It has been said by medical men, however, that the operation should not be done when there is suspicion of coronary thrombosis. I cannot appreciate the logic of this. The thrombosis certainly cannot be increased by the operation and, since the disease in any event is a critical one, the possibility of losing the patient during the procedure ought hardly to be considered a deterrent. In making the diagnosis, however, there should be, for scientific reasons, a sharp differentiation from the pain of syphilitic aortic disease including aneurysm. If syphilis is present, operation should be preceded by antisyphilitic treatment.

Surgeons who have operated in this condition appear by common consent to have selected the side of the most pronounced brachial pain as the one for the first operation.

In my first case, the patient, as stated, continues to have certain sensations in various parts of the thorax, head and arm. Others have noted paresthesia or pain in various locations, particularly in the ear and the angle of the jaw. Whether these sensations are of any clinical meaning we do not yet know, and it may require a large number of observations to establish any rules of incidence or cause. That any possible annoyance from these symptoms will ever act as a contraindication to the operation I cannot believe, for, in well marked angina pectoris, the suffering is so acute and fear inspiring that anything else seems preferable. All three of my patients were told before operation

that there would be a change in the facial expression from the ocular signs, but all three did not for an instance regard this as worth a moment's consideration. It is necessary, however, to warn the patient's that there will be some facial deformity. In one case, that of a 32 year old unmarried woman who had spent years in institutions because of severe spasmodic asthma and who was actually disabled, operation was refused because of probable Horner's syndrome. Still, asthma is not so distressing nor so dangerous as angina pectoris.[7]

DISCUSSION

DR. WILLIAM D. ANDRUS, Cincinnati: In speaking of sympathectomy, Dr. G. Carl Huber of Ann Arbor, who has done considerable work on the sympathetic, raises this question, Why is a cervical sympathectomy necessary? Why not simply section the trunk, or resect a short segment, below the inferior cervical and first thoracic ganglions? All the preganglionic fibers for the cervical sympathetic emerge from the cord in the upper thoracic region. Hence, a simple section of the sympathetic chain as mentioned above should include all these preganglionic fibers, and, so far as we know, the postganglionic fibers, whose cells are in the chain ganglions, cannot function independently of their preganglionic connections with the central nervous system. It is an interesting theoretical point.

DR. JOHN L. YATES, Milwaukee: It has been shown fairly definitely that the pain, the distress and the danger in angina are occasioned by coincidental hyperactivity of the heart and spasms in the aorta and coronary arteries. If it is desired merely to control the pain, section of the cervical sympathetic, as suggested by Dr. Andrus, will be effective. As far as giving that person a better chance for survival by reducing the danger of the attack and also lowering blood pressure, probably more has to be done. Cardio-accelerator fibers proceed from the superior, the middle and the inferior cervical ganglions. From the superior, thoracic ganglion fibers go to the aorta which deliver vasoconstrictor impulses. Pathways of sympathetic fibers are inconstant, and the variations are unknown. If mere sections are done in the cervical region, the pathways and impulses to the heart and to the larger vessels are not constantly if ever completely obstructed. Pain is inhibited. If the mere inhibition of pain is to be desired, well and good. If an attempt is to be made to give that person the maximum of physiologic benefit, the thoracic ganglion must also be extirpated in order to assure stopping undue cardiac acceleration and undue arteriospasm.

Another important point is emphasized in advocating doing the first operation on the left side, because, as Dr. Lilienthal's paper showed, a certain proportion of patients will not get relief if only one side is attacked. Bilateral cervical and superior thoracic sympathectomy may reduce blood pressure and does not affect general physical capacity. It therefore has a bearing not only in treating angina but also in relief of certain types of essential hypertension.

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7. Since this paper was written, I have had another case in which both sides were operated on with an interval. In Case 2, after a recurrence of symptoms, the right sympathetic chain was successfully extirpated. These cases will form the subject of a later report.

Dr. Charles H. Mayo, Rochester, Minn.: I believe that this operation is based on sound principle. Many years ago, when Jaboulay, and following him, Jonnesco, brought out the idea of cervical sympathectomy for the treatment of exophthalmic goiter, I tried it in a number of cases. It seemed to be too serious an operation compared with direct work on the glands. In several cases, I removed the middle and superior sympathetic ganglion on both sides. Then Jonnesco advanced the idea that epilepsy could be cured by the removal of the sympathetic ganglion. I presume the idea was that it worked about like circumcision and various allied procedures; most anything that is done for an epileptic is good for him for a while, but his epilepsy comes back.

For a good many years, Dr. W. J. Mayo and I operated at a large hospital for the insane here, and in another a hundred miles west of here, and I had the opportunity to apply sympathectomy to many cases of epilepsy. Permanent relief was not obtained in any case. The operation is not difficult. I always cut down on the superior sympathetic ganglion and then trace down to its middle. The superior sympathetic has as many little branches as a centipede has legs, and they seem to be hooked up to everything, but it is easy to trace them all.

I had a patient, a major in the United States Army, who, because of repeated attacks of angina pectoris, had been compelled to give up his work. He came to me, thinking he might have exophthalmic goiter. He could never climb half a flight of stairs without stopping to rest, and exertion brought on attacks of pain in the heart, a feeling of pressure in the chest, the superchest and the neck, and a feeling of pending death. I removed both sympathetics, that is the middle and superior on both sides, and wholly relieved his pain. This was done twelve years ago; he is still able to get about, and no longer has pain.

Dr. Frank C. Mann, Rochester, Minn.: I wish to discuss this interesting problem purely from the standpoint of the physiologist. As you know, the discovery of anesthesia and then, later, the pathology of disease and the application of surgery opened up the field of surgery, and we had a great advance in intra-abdominal surgery. That occupied the surgeon for a good many years, and then he gradually went into other fields until now we find the surgical invasion of the sympathetic nervous system. While that invasion has been slow, extending back in some cases over many years, it is more active in the last few years than ever before.

To a certain extent, the surgeon is perfectly justified in entering this field; the physiologists have been interested in it from a certain standpoint, mainly physiology; the pathologist has not been interested in anything he couldn't see or put under a microscope, and a lot of these things that one sees clinically one can't study that way. So it is left to the surgeon to try to take care of the condition, and, in doing so, he has invaded many fields, and in many of them he has preceded the physiologist, for instance, in the case of the gallbladder. He is doing the same thing in regard to the sympathetic nervous system, and we hope that from these experiments on human beings, being done at the present time, that we will enlarge our knowledge to a great extent in regard to the action of this very important physiologic mechanism.

There are two or three things, however, he must clearly bear in mind if he invades this field. First, in the disease of animals, we have studied the distribution and course of these fibers. I would hesitate very much to predict the course of any particular fibers of the sympathetic nervous system in any

animal that I had not studied, or that had not been studied by a competent observer, and so far we know very little as to the course of these fibers in the human being. The surgeon is doing operations and attributing them to sections of certain fibers, which we know, in all probability, are not at the place in which he is doing his surgery.

Surgeons have advanced and gotten ahead of both the physiologist and the anatomist, but one must not attribute the good results to the section of something which is not there. If one is going to get the cardio depressor nerve, one must section the vagus and not the sympathetic.

I think that, in justice to the patient, one should explain that it is an experiment, and carefully record the results so that we will know what effect these nerves have on the physiologic condition, and in that way we will advance faster than we will in the experimentation that we are doing on animals. Then one must remember when sectioning this nerve that one is only after a small group of nerves and one sections a large number. One must always bear in mind that, for the insistent ringing of a telephone bell in any building, it would not be very good economy to section the whole cable.

Dr. George J. Heuer, Cincinnati: In connection with Dr. Lilienthal's paper, I want to report on a case of angina pectoris in which my associate, Dr. Reid, operated about a year ago, and which is interesting in that it demonstrates what widespread neurologic changes may take place following cervical sympathectomy. This was a perfectly typical case of angina, and, at operation, Dr. Reid removed the superior, middle and inferior cervical ganglions, together with the cord connecting them. The patient made an uninterrupted recovery, and so far as the attacks of angina are concerned, he has been completely relieved. However, he came back to the outpatient clinic several months after the operation, complaining of pain in the face, and he was sent to me with a diagnosis of trigeminal neuralgia. When I came to examine him, his symptoms strongly suggested those of trigeminal neuralgia. Various sensory tests showed that his face was not totally but partially anesthetic, this partial anesthesia involving his tongue and the inside of his cheek. He also had disturbances in taste. In other words, he had the sensory disturbances not completely like but similar to those which follow division of the sensory root for trigeminal neuralgia. And when I came to examine him further, I found that he also had sensory disturbances over his left arm, extending to his fingers; he didn't appreciate touch or pain, and he did not have the ability to distinguish two points; he had inability to distinguish the form of objects in his hand. He also had sensory disturbances over his thorax to the costal margin.

This case of Dr. Reid's (J. A. M. A. **83**:114 [July 12] 1924) I bring up here as an interesting observation, with the hope that others will report similar cases in their experience. Those of us who have been interested in the condition have all had a patient or patients with trigeminal neuralgia who, when operated on, have failed to get relief which we expected. Their pain continues and in the distribution of the fifth nerve, even though, on examination, the face is totally anesthetic.

It would appear from Dr. Reid's experience that there may be an explanation for these irregular cases of trigeminal neuralgia; that they may, in other words, be examples of sympathetic ganglion neuralgia. I recently had the opportunity of seeing one of these cases, a man who has not been relieved of his pain by sensory root division. On pressure on the superior cervical ganglion, I was able to provoke attacks of pain which radiated in the dis-

tribution of the fifth cranial nerve and which were quite similar to the pain from which he has been and is still suffering. It is possible that a cervical sympathectomy may relieve his pain.

Dr. Willy Meyer, New York: I should like to mention that the resection of the superior cervical sympathetic ganglion was also proposed for treating glaucoma. About twenty years ago, I did this operation successfully on the affected side. No particular improvement of the disease of the eye was observed.

It certainly is of keen interest to see how the surgeon can improve some thoracic diseases by attacking the nerves as they pass along the neck. Besides the resection of the sympathetic in cases of angina pectoris, we all know that the phrenic nerve is often excised at the neck when operating for advanced pulmonary tuberculosis.

SUBPHRENIC ABSCESS AND ITS DIFFERENTIAL DIAGNOSIS ROENTGENOLOGICALLY CONSIDERED

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NEW YORK

In view of the many cases recently reported of difficulty encountered in the diagnosis of subphrenic abscess, resulting in delayed operative procedure, it appears desirable to stress the roentgen-ray diagnosis of this condition. Cottle [1] recently reported a case of subphrenic abscess following removal of the appendix and excision of a duodenal ulcer, in which the diagnosis was disputed for three and a half months. In spite of the fact that the clinical evidence indicated a chest lesion, the roentgenologist insisted that the fluid was beneath the diaphragm. After a delay of about four months, the roentgenologist's diagnosis of subphrenic abscess was confirmed by aspiration and, later, by operation. Case 1 (Figs. 1-6) presented somewhat similar difficulties.

The physical signs apparently are often misleading. In a recent paper by Douglas,[2] the following interesting explanation was offered:

> As usually described, the physical signs found are dulness or flatness, diminished breath and voice sounds and vocal fremitus, with the presence of râles over the base of the lung. The area of dulness is characterized by being convex upward and not changing its area with a change of position of the patient. When gas is present in the abscess cavity, there are three zones of different resonance on percussion; normal above, tympany caused by the gas below this, and the area of flatness caused by the pus, which is continuous with the liver dulness on the right side. When there is also fluid in the pleura, an area of flatness forms a fourth zone between the pulmonary resonance and the tympanitic zone caused by the gas beneath the diaphragm.
>
> These are the classical signs recorded and would probably be present if each layer or zone were superimposed on the other evenly like a pile of books or a layer cake. But they are not, and frequently the physical signs become most confusing.

Although the physical signs of subphrenic abscess resemble closely those of pneumonia or fluid in the pleural cavity, a careful roentgen-ray study will differentiate between those conditions. Douglas says, "Roentgenographic study with the fluoroscope and films is the best method not only of confirming or making the diagnosis but also of determining the position and size of the fluid accumulation."

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1. Cottle, G. F.: U. S. Nav. M. Bull. **19**:683 (Nov.) 1923.
2. Douglas, John: Ann. Surg. **79**:845 (June) 1924.

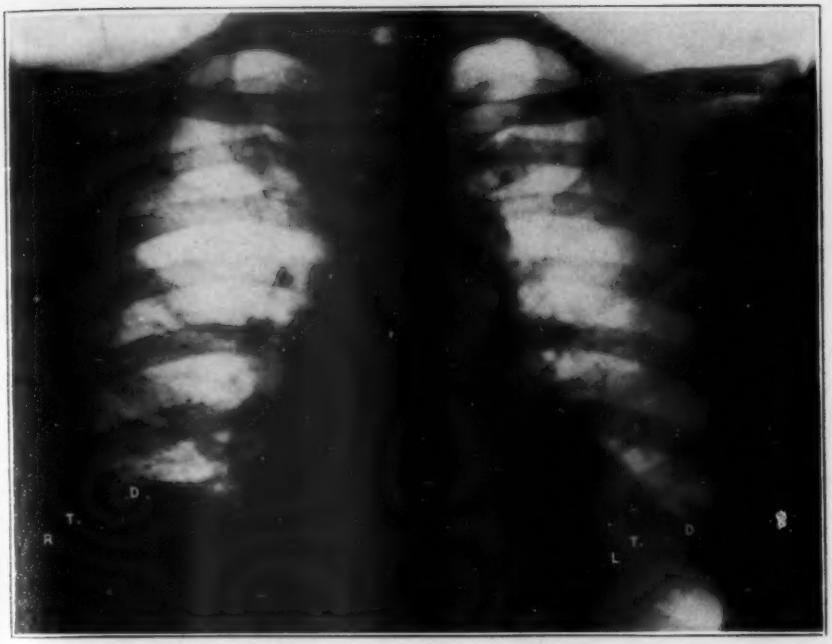

Fig. 1 (Case 1).—Subphrenic abscess one day after onset of acute right sided abdominal symptoms; absence of pleural involvement.

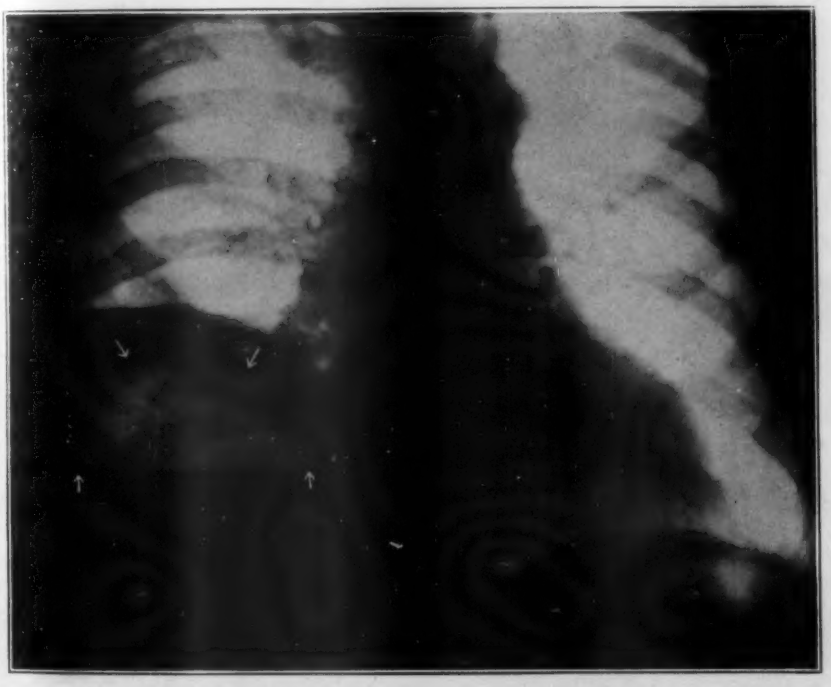

Fig. 2 (Case 1).—Subphrenic abscess, clinically diagnosed as "chest abscess," four weeks later than Figure 1; exploratory puncture; pleural involvement. Upper arrows indicate location of diaphragm; lower, fluid level in subphrenic abscess.

From a roentgen-ray standpoint, these cases may be divided into two classes; first, those in which gas is present together with a fluid accumulation (Case 1, Figs. 1-6) and, second, those in which there is a fluid accumulation without the presence of gas (Case 2, Figs. 7-10). Both types have in common elevation of the right side of the diaphragm. In determining this point, however, it is necessary to take into account the relatively higher position of the right side of the diaphragm in the normal individual. And, conversely, one should never be misled by the

Fig. 3 (Case 1).—Subphrenic abscess; lateral exposure distinctly shows abscess cavity to be beneath the right diaphragm; shadow of posterior portion of right diaphragm over spine. Comparison should be made with normal (Fig. 4).

fact that, in certain healthy persons the left side of the diaphragm may be as high as the right, or in cases of eventration, the left side of the diaphragm may reach even a higher point than the right side. In rare instances there is a bilateral eventration of the diaphragm. While a collection of fluid beneath the diaphragm on the left side has not been

personally observed, it may occur and cast a shadow between the diaphragm and the gas in the cardiac end of the stomach and splenic flexure of the colon.[3]

At times, perforation of a hollow viscus may occur, without the presence of free gas beneath the diaphragm, a few hours later (Fig. 11).

In persons suffering from marked enlargement of the liver due to abscess, new growth, or, rarely, to extensive interstitial hepatitis, the right side of the diaphragm may be relatively higher than the normal.

Fig. 4.—Lateral exposure showing normal relative positions of right and left sides of diaphragm, for comparison with Figure 3.

A smooth elevation of a portion of the diaphragm, particularly of the mesial portion, may be of no pathologic significance. This appearance may be due to unusually thin structure or to irregular contraction.

The extreme variation in height of the diaphragm between inspiration and expiration might lead also to an error in interpretation. This

3. Wessler and Jaches: Clinical Roentgenology of Diseases of the Chest, Troy, N. Y., Southworth Company, 1923, pp. 489-499.

is particularly true in the case of critically ill patients when the exposures are apt to be made in expiration, at which time the diaphragm is at its highest point, even though the patient has been requested to hold a deep breath. Furthermore, these patients are usually examined roentgenographically in the dorsal or prone position, in either of which the diaphragm is forced upward by the abdominal contents.

To gain the maximum information, it is necessary to examine the patient in at least two planes, one of which should be the upright, if

Fig. 5 (Case 1).—Subphrenic abscess two weeks after thoracotomy, which revealed a very small amount of exudate in the pleural cavity without free pus; position of probe passed through thoracotomy wound. Two weeks later, the cavity was evacuated following exploration by a needle which was directed *downward* through the diaphragm. Arrows indicate fluid level in subphrenic abscess.

possible. When the patient is too ill even to be turned, in addition to a dorsal exposure it is always possible to take a lateral exposure by placing the film against the patient's right side and passing the ray through the body from left to right. In this way, a fluid level might be demonstrated in cases in which there was air.

Few patients are so ill that they cannot be turned on the side, in order to make a straight lateral exposure which will show the costophrenic space. While the patient is on his left side, an exposure should be made with the film against the front of the chest and abdomen and the ray passing through the body from behind forward. This procedure may be reversed by placing the film against the patient's back and passing the ray in an anteroposterior direction (Figs. 8 and 9). It is desirable to have both these if possible. Added information may be

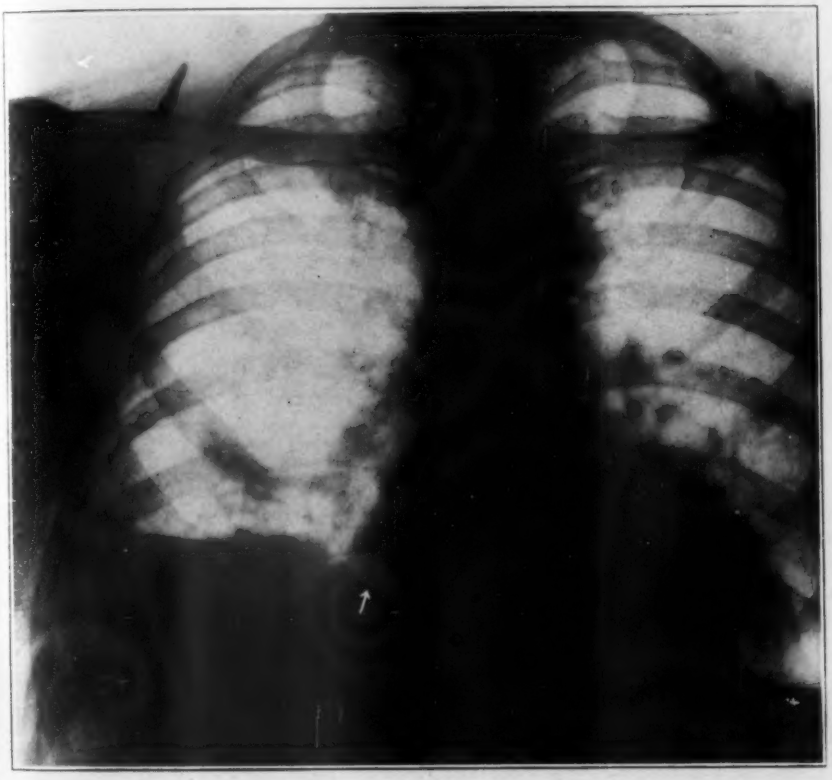

Fig. 6 (Case 1).—Five weeks after drainage of subphrenic abscess. Diaphragm is returning to normal position. Subsequent roentgen-ray examination seven months later showed only slight elevation of right side of diaphragm with some limitation of motion.

obtained by repeating this procedure with the patient lying on the right side. These lateral exposures are of particular value in those cases in which it is difficult to differentiate between a small collection of fluid above or below the diaphragm.

When the patient is able to sit, an examination should always be made in the upright position, first, with the ray passing from behind

Fig. 7 (Case 2).—Subphrenic abscess (all roentgenograms of Case 2 were taken at the bedside) showing elevation of right side of diaphragm, twelve days after onset of acute abdominal symptoms, supposedly due to perforation of a peptic ulcer, and four days after incision and drainage without rib resection; absence of pleural involvement.

forward to demonstrate a possible fluid level (Figs. 2 and 5), and, secondly, with the film against the right side and the ray passing from left to right to show the costophrenic space (Figs. 3 and 4).

In unusual cases, exposures could be made in an extreme Trendelenburg position in order to determine the lower limit of the abscess cavity.

Careful study of exposures made in these various positions will, in itself, usually afford sufficient evidence to render possible a diagnosis of subphrenic abscess in a typical case, in spite of confusing physical signs.

In a case of suspected subphrenic collection, exploratory puncture does not appear to be advisable because of the danger of infecting the uninvolved pleura which is reached through the costophrenic space. Whereas a complicating pleuritis effusion is not unusual, we have observed at least one case in which there was no roentgenographic evidence of involvement of the pleura until after the exploratory puncture had been made and purulent material withdrawn from the subphrenic abscess.

In differentiating empyema from subphrenic abscess, utilization of the lateral position will aid greatly in determining the presence or absence of fluid in the costophrenic space. The diaphragm will be in a relatively normal position in empyema, while in a subphrenic abscess it will be elevated (Figs. 3 and 4). Even in encysted collections of fluid in the chest cavity, the position of the diaphragm, being relatively unchanged from the normal, affords the means of making a differential diagnosis from subphrenic abscess in most cases.

The position of the diaphragm being the criterion by which the diagnosis of subphrenic abscess is made, all methods must be resorted to to determine this fact. If the foregoing methods fail to give the information desired, it may be necessary to resort to injection of air into the peritoneal cavity, as recommended by Dr. W. H. Stewart. It is reasonable to believe that this procedure is at least as safe as exploratory puncture and gives more definite information as to the exact location of a suspected abscess cavity beneath the diaphragm.

Conversely, when the evidence is more suggestive of a collection of fluid above the diaphragm, injection of air into the pleural cavity followed by roentgenographic examination in various positions might be advisable. Free fluid in the pleural cavity would at once become evident by the production of a fluid level. An encysted collection might become much more sharply outlined and separated from surrounding structures, particularly from the diaphragm.

CONCLUSIONS

1. Subphrenic abscess can be differentiated roentgenologically from other diaphragmatic lesions.

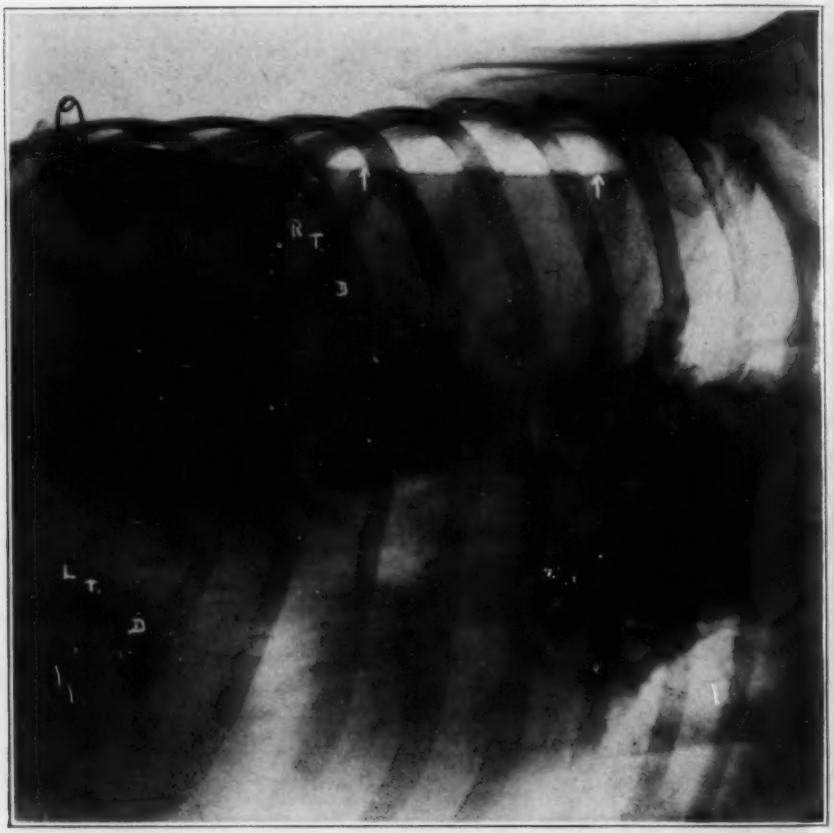

Fig. 8 (Case 2).—Subphrenic abscess with complicating empyema five weeks after first subphrenic drainage; patient on left side, ray passing from front to back.

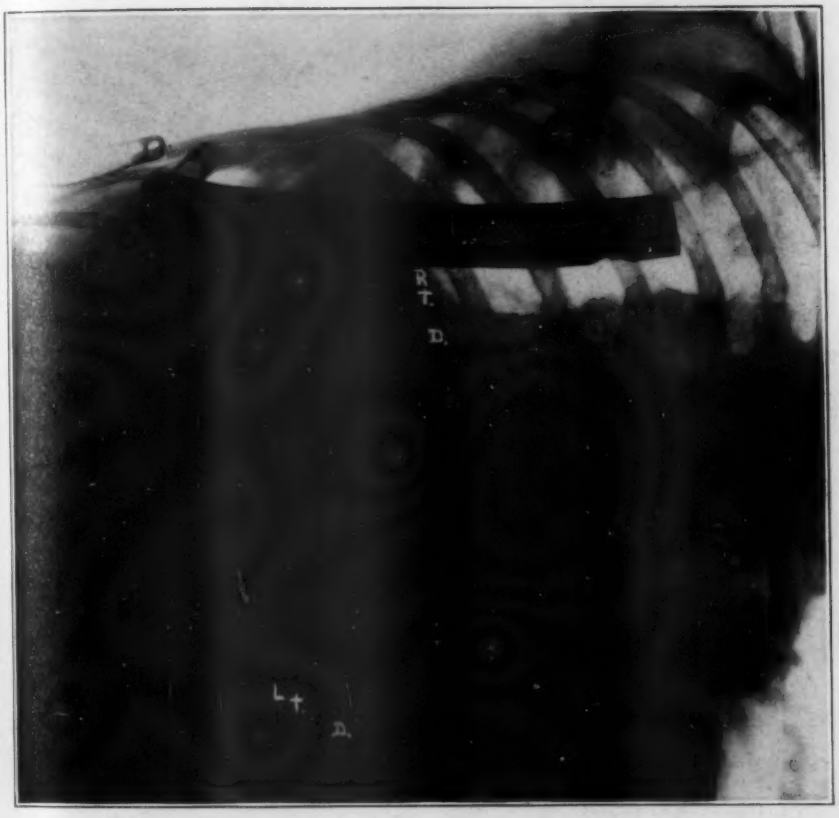

Fig. 9 (Case 2).—Subphrenic abscess with complicating empyema three weeks after rib resection for chest drainage; in spite of the excellent drainage, the lung failed to expand completely.

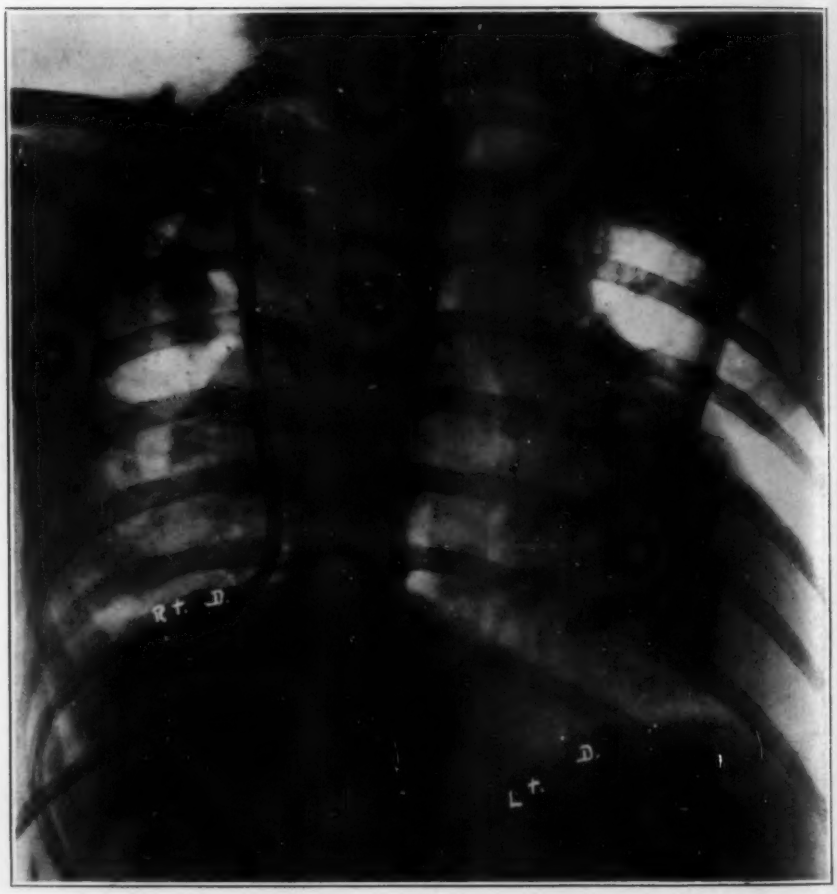

Fig. 10 (Case 2).—Subphrenic abscess with complicating empyema six weeks after rib resection; chest still draining; abdominal condition improved. The patient died three weeks later from gangrene of the lung.

Fig. 11.—Perforation of peptic ulcer *without* the presence of free gas beneath the diaphragm six hours after opaque meal and four hours after perforation. The perforation was sutured two hours later without gastro-enterostomy. Six months later, roentgen-ray examination showed excellent function of stomach.

2. Roentgenograms must be made in various positions in order to detect a shifting of fluid when gas is present. This can be accomplished with a bedside apparatus by moving the apparatus instead of the patient.

DISCUSSION

DR. NATHAN W. GREEN, New York: The differential diagnosis of subphrenic abscess as compared to pleural suppuration is apparently as difficult as Dr. LeWald's presentation of it is simple. It is a subject for much heated discussion when brought up in medical meetings, and I think all of us feel that we are not entirely sure whenever we encounter one of these conditions.

The roentgen ray, as you will see from Dr. LeWald's clear presentation, is the greatest help in the world in making the differential diagnosis.

The anteroposterior roentgen-ray examination of the patient either sitting up or lying on his side seems to be essential. The fluid level with gas above it and the whole shadow below the diaphragm leads us to make the diagnosis. If we do not get the level and are still in doubt, I think that Dr. LeWald's suggestion that Dr. Stewart's procedure of making a pneumoperitoneum is perhaps less dangerous and more efficacious than continued and persistent using of a needle in the region in order to find pus; because, as has been demonstrated in one of these cases, when pus is found, one doesn't know whether it is above or below the diaphragm.

DR. GEORGE A. STEWART, Baltimore: The typical roentgenographic signs of a subphrenic abscess are the presence of a high diaphragm with gas bubble and fluid level below.

These signs are not always present, however, and in many cases it is difficult to recognize the diaphragm. The left side can be made out by inflating the stomach with air or giving the patient a full barium meal; the right side is more difficult. In fact, unless the patient is extremely ill it is advisable to inflate the peritoneal cavity with oxygen, by which method one can easily recognize the right side of the diaphragm.

THE VALUE OF LATERAL AND OBLIQUE ROENTGEN-LIGHT EXPOSURE IN THE DIAGNOSIS OF THORACIC AFFECTIONS *

J. S. PRITCHARD, M.D.

BATTLE CREEK, MICH.

What information may be obtained from an oblique roentgen-ray study of the thorax?

1. The location and estimation of the extent of substernal enlargements.

2. The location and outline of mediastinal tumors.

3. The size and outline of the glandular tissue (bronchial and thymus).

4. The location of foreign bodies of a certain type (Fig. 1).

5. The outline of the exact course of the trachea with the detection of any irregularity in its size, shape or position.

6. The condition of the thoracic spine (Fig. 2).

7. An estimation of the size, shape and density of any part or parts of the thoracic circulatory system (Figs. 3, 4 and 5).

8. The condition of the diaphragm and the location of posteriorly formed pleuropericardial or spinal pericardial adhesions (Fig. 6).

9. The location and outline of pulmonary suppurations (the patient lying on the side).

10. The behavior of the "barium mixture" in its passage through the esophagus.

11. The location of calcareous deposits, such as calcified glands, below but within the circle of the aortic arch. These conditions sometimes cause pain simulating that caused by angina pectoris (Fig. 7).

Why is that portion of the thorax lying behind the heart and basal vessels not investigated with more frequency?

1. The territory is inaccessible.

2. The boundaries are difficult to visualize.

3. The physical signs of abnormalities in this region are few.

4. Abnormalities occur with less frequency in this secluded field.

5. This area is not included in the average routine examination of the thorax, and is investigated only when the internist on physical examination, or the roentgenologist on viewing the anterior chest films, discovers something which suggests the advisability of an oblique visualization.

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* From the Department of Pulmonary Diseases, Battle Creek Sanitarium.

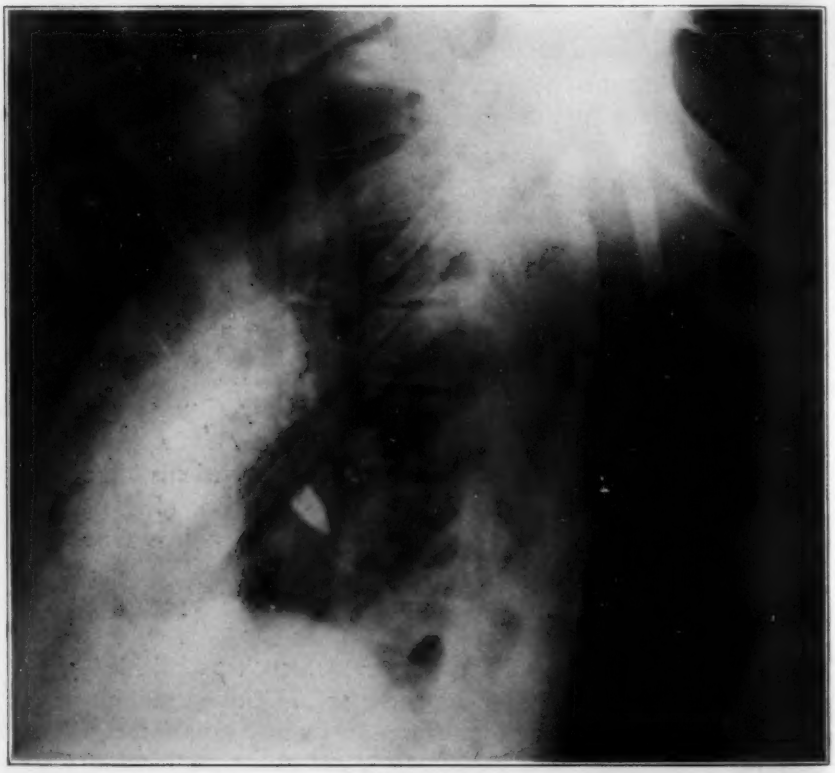

Fig. 1.—Metal head of lead pencil, seen on right oblique exposure, in the left bronchus where it had remained for twelve years, later successfully removed by Dr. Chevalier Jackson.

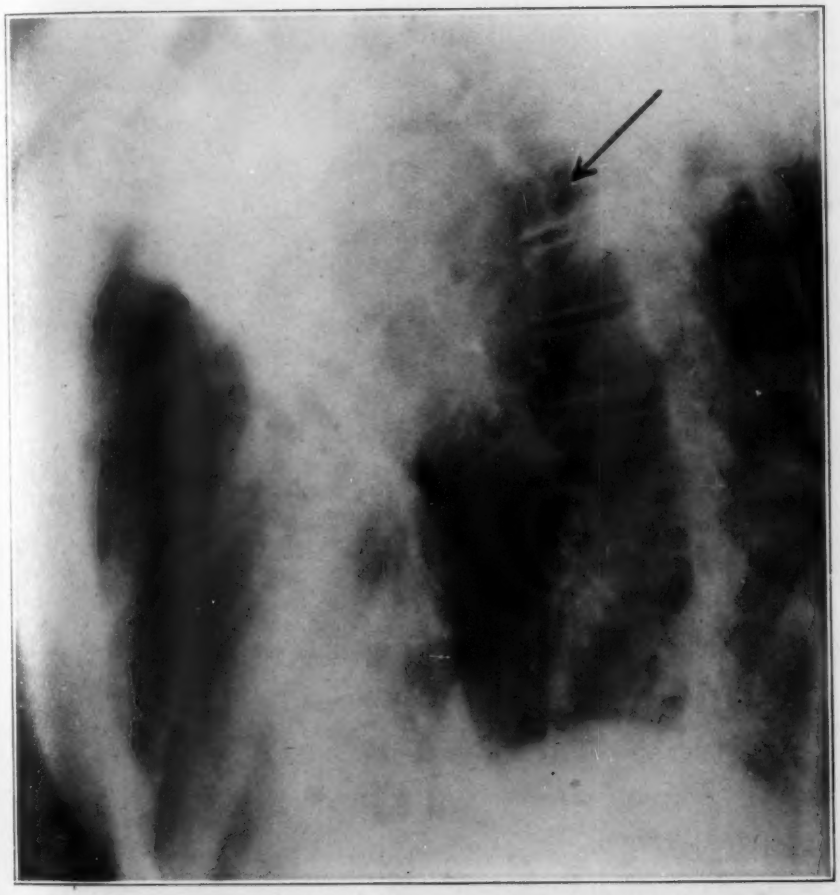

Fig. 2.—Fifth dorsal vertebra partly destroyed by pressure atrophy due to action of large aortic aneurysm.

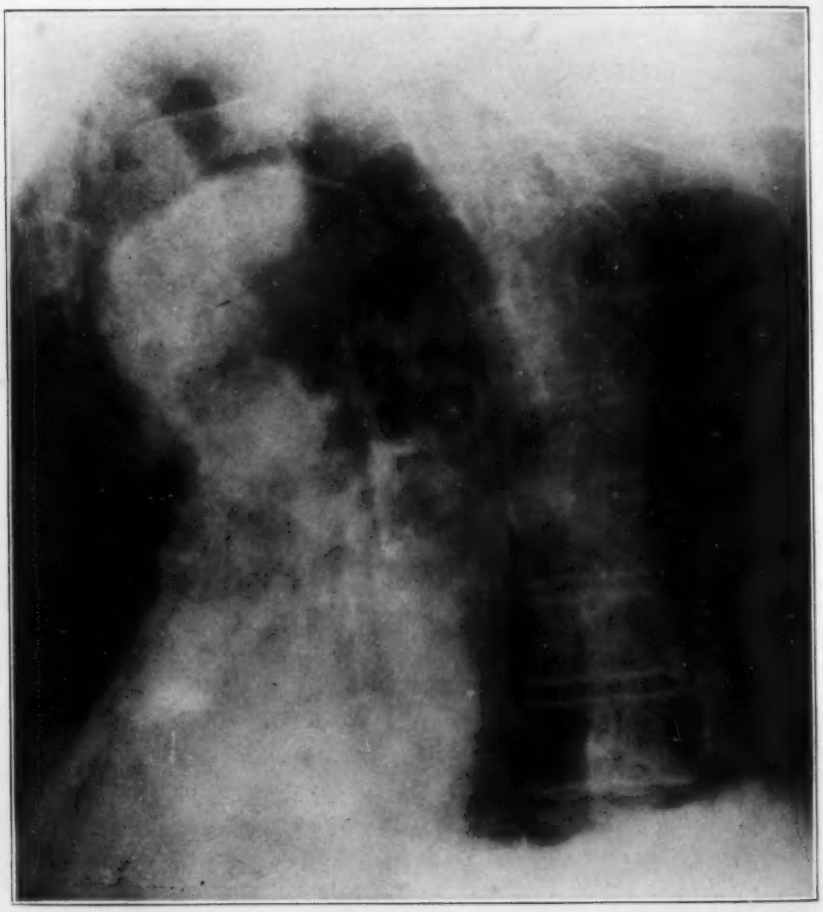

Fig. 3.—Right oblique exposure showing an extensive calcification of the walls of the entire thoracic aorta.

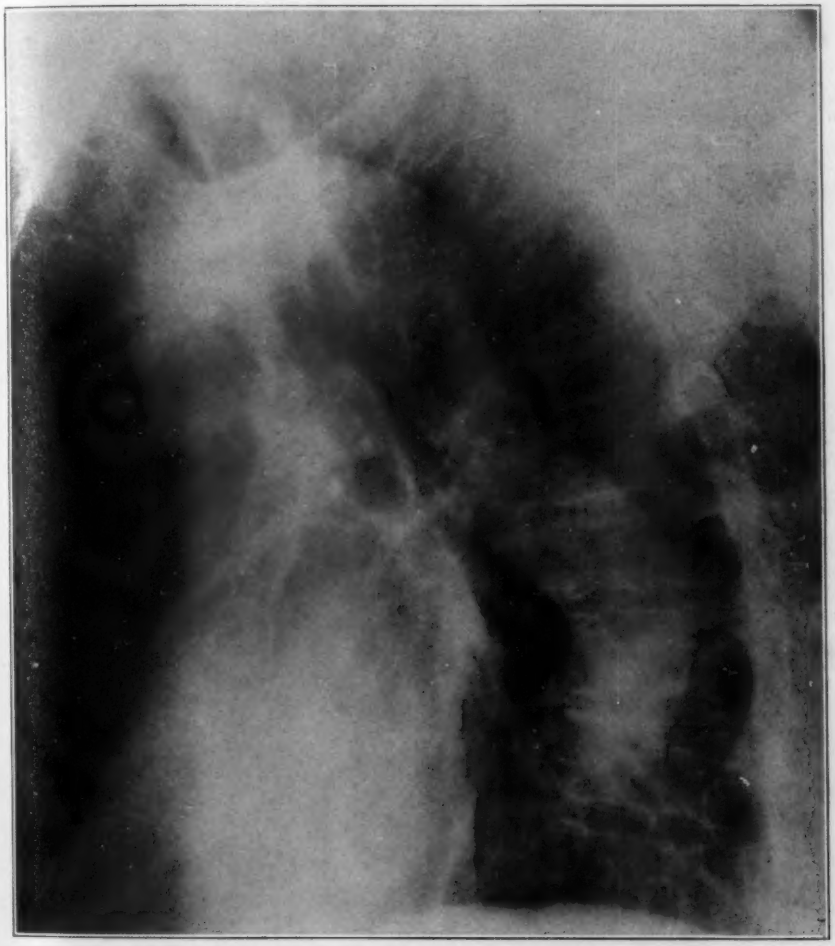

Fig. 4.—Distinct visualization of a tortuous thoracic aorta, not suspected on the posteroanterior exposure.

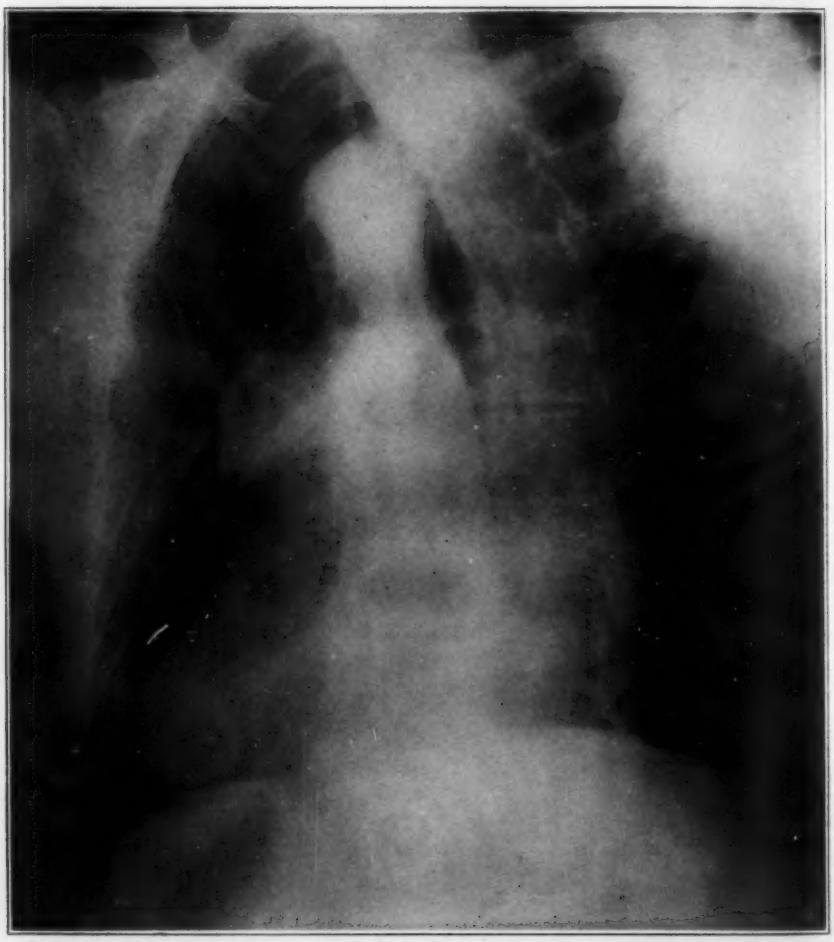

Fig. 5.—Localized, one-sided aneurysmal sac in the descending aorta, not seen in the postero-anterior position.

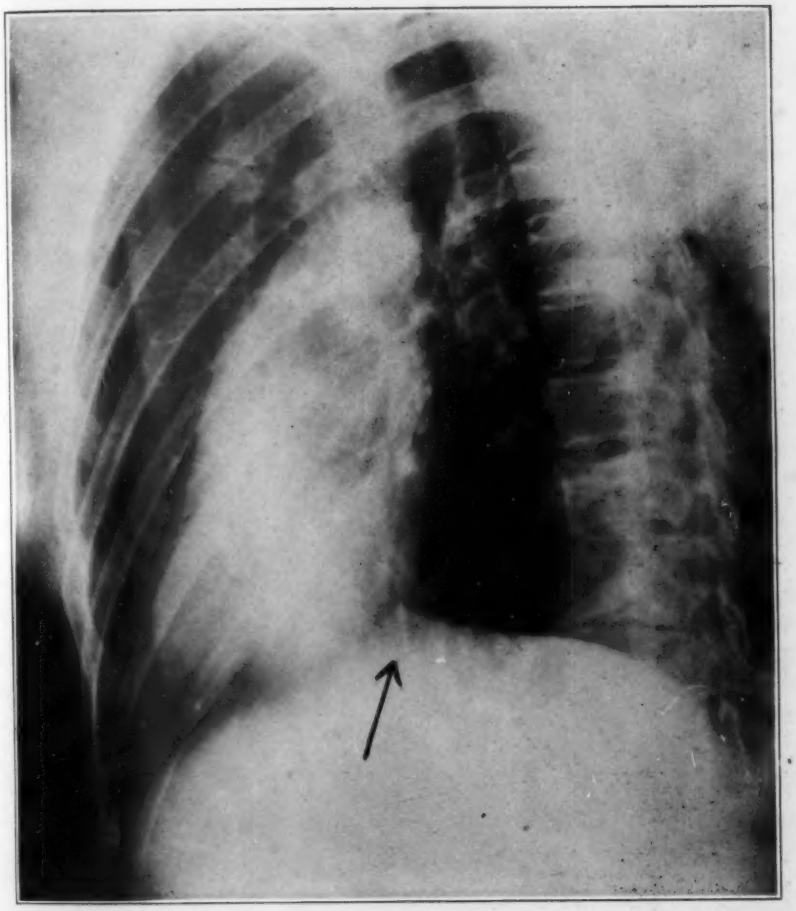

Fig. 6.—Pleuropericardial adhesions, seen on oblique exposure. which the postero-anterior position did not reveal.

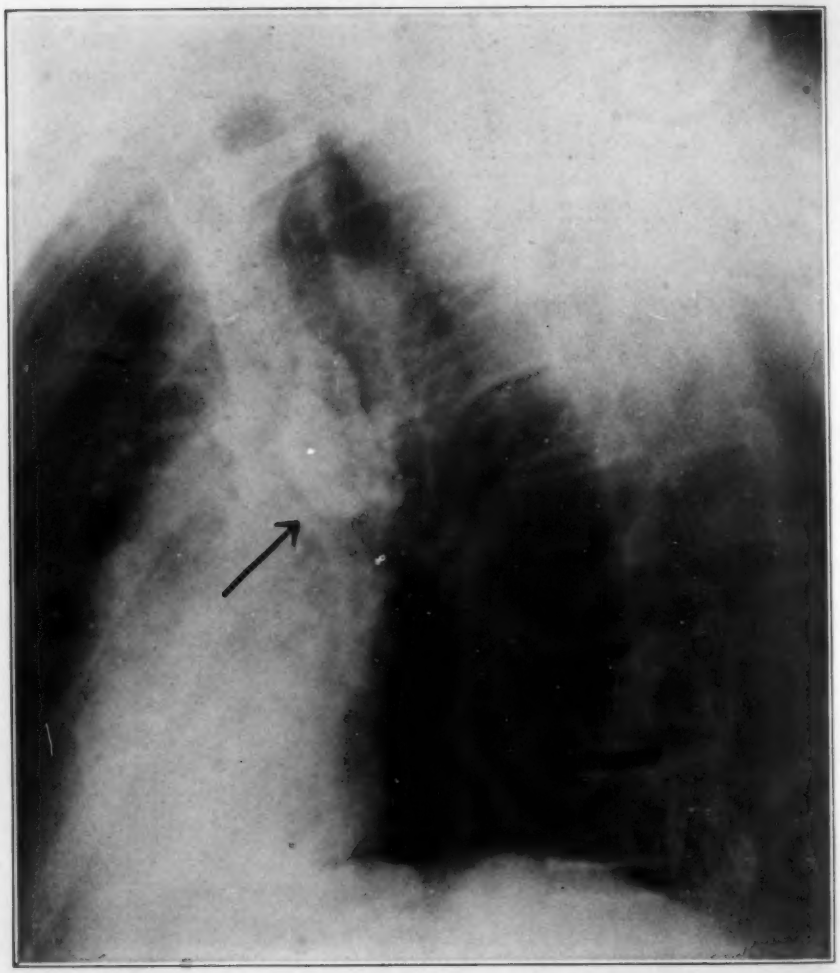

Fig. 7.—Calcified mass under aortic arch; the patient complained of chest pains simulating angina pectoris.

Fig. 8.—Correct position of patient against film in making a right oblique or lateral exposure of the mediastinal space.

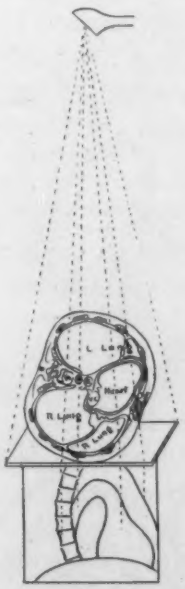

Fig. 9.—Penetration of the rays in the right oblique position (right chest to film).

What is the method of procedure in the study of the posterior mediastinum?

The history taking, a physical examination and the roentgen-ray study. The latter method must be relied on to give the most valuable information. In studies of the oblique position, the fluoroscope will not reveal all that the roentgenogram can visualize and should not be relied on except as a registrar of movement, such as the abnormal contours of an adherent diaphragm during deep inspiration. The passage of an opaque diagnostic medium, like barium, down to the esophagus is another example.

ROENTGEN-RAY TECHNIC

A right anterior oblique chest film exposure may be obtained as follows: The patient should place the right side of the chest in apposition to the film. The tube target should be set at a distance of 1 meter behind the patient and opposite the middle of the left posterior axillary line. The time used in exposure is from three-eights to one-half second with a 30 milliamperage. The amount of penetration required is from 7 to 8 kilovolts. The degree of obliquity can be obtained with reasonable accuracy after a few trials. The line of penetration of the rays is more of a lateral angle than a true oblique (Fig. 9). This exposure gives an excellent outline of the heart and aorta (Figs. 3 and 4).

A left anterior oblique chest film exposure is obtained by the patient's placing the left side of the chest in apposition to the film while the rays penetrate the right middle posterior axillary line. This exposure, in turn, gives a good view of the trachea and its bifurcation. Tumors or enlarged glands in this region are best studied by the left anterior oblique procedure.

SUMMARY

These conclusions are based on the evidence compiled by a clinical study of over 12,000 patients sent to my department on account of symptoms suggesting the existence of some lower respiratory disorder. The clinical study, including laboratory methods, was in each case augmented by stereoscopic films of the chest. Regardless of this procedure, we failed, in many instances, to reveal the cause of the symptom complex. This fact led us to adopt as a routine method, in all cases coming to the chest department, the exposing of the posterior mediastinum by oblique roentgen-ray study. At the present time, some 3,000 oblique roentgen-ray exposures have been made. We feel amply rewarded for including this procedure in the general study of suspected intrathoracic affections.

DISCUSSION

DR. A. L. LOCKWOOD, Toronto: It was due to the work of Dr. Pritchard that we were stimulated, a year ago, to take up oblique roentgen-ray studies of the contents of the posterior mediastinum. When we first started, we thought we would make oblique roentgenograms in questionable cases, but after we had employed their use in about thirty cases, we found that we were not able to interpret the findings; so we had to start taking them as a routine in normal cases. We made oblique roentgenograms of forty normal people who had no chest lesion whatever, and we were absolutely bewildered by the findings we got in the first fifteen or twenty of these.

Now, however, after making these roentgenograms for a year, and by a careful study of them, we have made some extraordinary observations of conditions that we did not suspect, particularly in such pictures as some of these that Dr. Pritchard has shown. In some of these, in which the lungs are apparently clear and the heart shadow does not appear to be increased, there is possibly just a certain displacement of the aorta or extra little bulge around the right or left border of the heart which we cannot account for. Very often the oblique ray shows the shadow to be due to enlarged mediastinal glands or an early growth.

I am not yet in a position to express an opinion of what value these oblique roentgen-ray exposures are going to be, but we ourselves are thoroughly satisfied that the making of oblique roentgenograms should be routine in every questionable case, and we are not quite certain that we will not take them as a routine in all our thoracic cases.

DR. J. J. SINGER, St. Louis: I saw Dr. Pritchard's work at Battle Creek, a year ago, and the beautiful pictures he showed today. Working as I do in St. Louis along the same lines that he does in Battle Creek, I probably appreciate this work more than others who are not living, as it were, in the dark room. I have always felt that it is just as logical to interpret a picture taken anteroposteriorly and give a final opinion as it is for a physician to examine the anterior part of the chest and express his opinion. It can't be done. The greatest mistakes we have made have been made by taking a plate in one direction and expressing what we thought was present.

I find in reports of roentgen-ray exposures from the roentgenologic department that wide aortic aneurysms are diagnosed very frequently. The measurements are taken across the upper mediastinum and of the length of the heart, and so forth, and aneurysm is diagnosed by the width of the mediastinal shadow. If that patient were merely turned a fraction of a degree, we could readily see that the aorta is perfectly normal and that the shadow represents thickened hilums or enlarged glands. This method aids a little in the study of the mediastinum: Not only should the patient's right side be turned to the plate with the left shoulder forward but also the patient should be bent over at an angle of 40 or 50 degrees, in order that one may still further expose the mediastinum.

ETHYLENE AND PRELIMINARY MEDICATION

JAMES T. GWATHMEY, M.D.
NEW YORK

There are fourteen hydrocarbon gases, varying in value from acetylene to octane.

All these gases have anesthetic value, but most of them have other properties that preclude their use in surgery. For instance, with acetylene there is always the possibility of explosion, although clinically its anesthetic value seems to be higher than ethylene and the others in the series. Propylene is easier to handle and administer than ethylene. If the objectionable odor and other impurities could be eliminated, it might possibly prove a valuable anesthetic agent; but as my laboratory experiments with propylene and three other hydrocarbon gases led to no conclusions definite enough to be of practical value, this paper will be confined entirely to ethylene and its different methods of administration.

HISTORY

Experiments with ethylene by different men at various periods bore no practical results or lasting benefits to mankind until the present era. Luckhardt [1] deserves the credit for placing ethylene on a firm scientific basis and establishing it as a safe anesthetic agent. Brown [2] read a paper before the Academy of Medicine, Toronto, Feb. 20, 1923, on "Experiments with Ethylene as a General Anesthetic," thus clearly establishing priority, since the first paper of Luckhardt in the *Journal of the American Medical Association* did not appear until over one month later, i. e., March 17, 1923. Luckhardt experimented on laboratory animals, in 1918, and, in December, 1922, sent to the secretary of the American Physiological Society the title of a paper dealing with ethylene anesthesia, but for some reason the paper was not put on the program even by title. Luckhardt evidently commenced work on ethylene before Brown, and the merest accident prevented priority of publication. Unfortunately, no medical publication anywhere distinguishes between articles dealing with an entirely new and original discovery and a rehash of effete material. Brown must therefore be given full credit for originality, independent research, and priority of publication.

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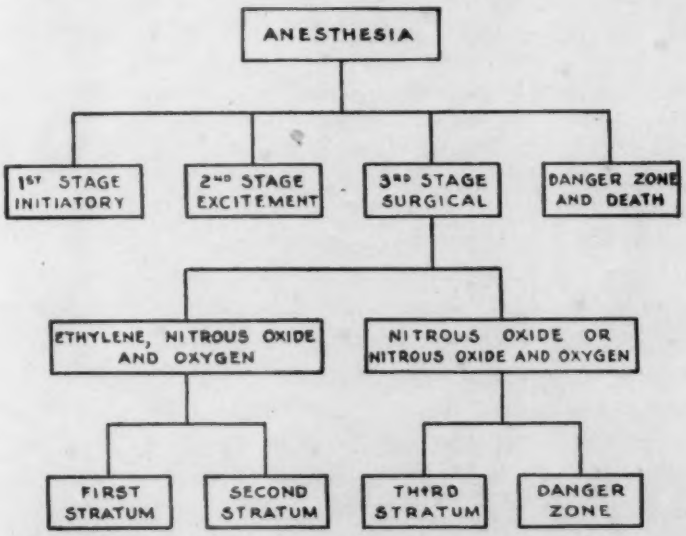
1. Luckhardt, A. B., and Carter, J. B.: Physiologic Effects of Ethylene, J. A. M. A. **80**:765-770 (March 17) 1923.
2. Brown, Easson: Canad. M. A. J. **13**:210, 1923.

Cotton's [3] work, placing ethylene in ether and antedating both Luckhardt's and Brown's experiments, while possibly having little or no bearing on the development of ethylene as an anesthetic per se, must not be forgotten. Cotton's ether has not stood the test of clinical trial, but his work has a practical bearing on one of the methods of administering ethylene.

LABORATORY AND CLINICAL

It has been demonstrated beyond all question, both in the laboratory and clinically, that ethylene is a safer and better anesthetic than nitrous oxid.[4] It is safer because, even without preliminary medication, the



The reason why ethylene is safer than nitrous oxid.

patient can be kept in the second strata of the third or surgical stage of anesthesia, pink, and separated from the danger zone by one strata as compared to nitrous oxid with the patient in the third strata, cyanosed and immediately bordering on the danger zone. As far as immediate safety to life is concerned, neither agent is as safe nor as good a relaxant as ether. Except for the nausea and vomiting, with resultant increase in wound and gas pains that usually follow ether by inhalation, it would remain the premier anesthetic.

However, it should not be forgotten that ethylene forms a cherry red hemoglobin derivative which may mask the important danger sign,

3. Cotton: American Year Book Anesthesia and Analgesia, McMechan, 1917-1918, pp. 153-164.

4. Luckhart and Carter (Footnote 1). Gwathmey, J. T.: Am. J. Surg. (Anesthesia Supp.) **38**:37 (April) 1924.

cyanosis. For this reason among others, ethylene cannot be *pushed*, as the usual danger signals are not apparent. Furthermore, it may be impossible, if respiration ceases, to revive by artificial respiration. When the gases are insufficient to relax the muscles, Dr. Herb switches to ether, which is the safe and proper procedure.

As compared to nitrous oxid, ethylene is safer, because analgesia comes on as consciousness is lost; with nitrous oxid, analgesia is never obtained in some patients except with accompanying asphyxia. Certainly, more vomiting follows the administration of ethylene than nitrous oxid, but never the projectile vomiting and nausea that occurs with ether. An animal or a patient is more quickly and easily resuscitated after respiratory arrest with ethylene than nitrous oxid. With ethylene, the patient does not usually perspire, thus conserving vitality.

EXPLOSIBILITY

The relative explosion hazard, for all practical purposes, is the same as that of ether, and depends in a very great measure on the oxygen content. In the office of M. Ecker (a dental and oral surgeon), before the introduction of ethylene, an explosion occurred involving the oxygen tank. Since the introduction of ethylene, and in the use of this anesthetic in over 4,000 administrations, no such accident has occurred. The accident in a Baltimore hospital was probably an oxygen-nitrous oxid explosion, but, as ethylene had been used previously, this agent was very naturally blamed for the accident. I myself have had two oxygen-ether explosions, but no accidents since using ethylene.

ETHYLENE VALUE

In anesthetic value, ethylene in many ways seems to fall between nitrous oxid and ether.[5] In my laboratory work, ethylene apparently affected the brain differently from nitrous oxid, as indicated by the bulging eyes with death from ethylene. This does not occur with nitrous oxid. If this is true, we here have an indication for using the two gases in combination. In my experience, a more elastic and safer anesthesia is obtained in using the triple combination than with ethylene and oxygen alone. Furthermore, in spite of improvement in purification since its first introduction, there is an objectionable odor to ethylene which, for the patient, is entirely avoided by using nitrous oxid and oxygen until unconsciousness ensues, and then adding ethylene as required instead of ether. Lundy was the first to use as a matter of routine carbon dioxid with nitrous oxid, ethylene and oxygen; it is a powerful respiratory stimulant. With a bag one-third

5. Lundy, J. S.: Northwest Med. **23**:126 (March) 1924.

full of carbon dioxid and two-thirds full of oxygen, it is not only useful in emergency surgery, but can also be used in obstetrics to resuscitate the new-born.

IN THORACIC SURGERY

In order to make these fundamental facts of practical value to thoracic surgeons, it is only necessary to refer to our laboratory and clinical work in France during the World War.[6] After various agents and methods were tried, it was found that an animal or patient was in the best possible condition for thoracic surgery when analgesic from preliminary medication, and unconscious only from nitrous oxid and oxygen. Previously, Marshall had found that while the blood pressure was sustained equally as well under ether vapor as with nitrous oxid and oxygen, there developed a few hours later a lowered blood pressure that did not obtain when nitrous oxid and oxygen were administered. Cannon [7] found that inhalation ether in a shocked animal caused a fall of blood pressure which might be as great as 30 or 40 mm. of mercury, whereas the same degree of anesthesia with nitrous oxid and oxygen caused no fall of blood pressure whatever. Our technic is to administer morphin in one-eighth grain (0.008 gm.) doses and repeat twice at twenty minute intervals, if no idiosyncrasy develops. The average for each patient is three-eighths grain (0.024 gm.). With this preliminary, we use from 15 to 30 per cent. oxygen with the nitrous oxid; and with the patient unconscious and pink, all reflexes are abolished and he is as safe and certainly far more comfortable than with local, spinal, paravertebral, or any other method of analgesia or anesthesia, moreover, with this added advantage, as stated by Yates, "positive pressure control made examination of the lung and operation much easier and eliminated the necessity for dangerous traction. It offered a simple test of the air tightness of the closure and of satisfactory hemostasis; the necessity for undue haste was eliminated." These remarks referred especially to soldiers shot through the lungs. The one danger from this form of anesthesia is overlapping. If the same degree of anesthesia is maintained with as without the preliminaries, the patient is immediately plunged into the danger zone. For conditions in which slight positive pressure is not indicated, local, spinal, or paravertebral analgesia may be used. I myself have used ether colonically for a very extensive cancerous condition, involving the removal of the chest wall. The patient's pulse jumped from 72 to 84 as the lung was exposed, but returned to 72 shortly afterward with no further indication of shock either during or after the operation. I consider

6. Gwathmey, J. T.: M. Rec. **97**:987 (June 12) 1920.

7. Historical Record of the Services of the Laboratory of Surgical Research. American Army at Dijon, 1918.

this as safe as the other methods just mentioned. For thoracic surgery, my personal preference is analgesia with unconsciousness, using preliminary medication and ethylene, nitrous oxid and oxygen.

PRELIMINARY MEDICATION

I wish to stress the fact that the preliminary medication[8] is of equal importance with the inhalation agent. Proper preliminary medication means the difference between life and death. I have had two deaths under nitrous oxid and oxygen when my opinion was ignored regarding preliminary medication. In one case, it was withheld because there was no elevator in this particular sanatorium. In the second case, one involving the thorax, during examination, with the patient lightly anesthetized, the eye and lid reflex active, the patient suddenly commenced very rapid breathing; and though oxygen was administered in plenty of time, he died. I am convinced that both these patients would be alive today if sufficient preliminary medication had been given to procure a complete brain block in connection with the inhalation anesthetic. The value of preliminary medication is not a matter of opinion but a matter of fact, as proved both in the laboratory and clinically. In the laboratory, Wallace proved that with preliminary medication the "margin of safety" (i. e., the difference between complete anesthesia and respiratory failure, using a 7 per cent. ether intravenously) was more than doubled both as to time and the amount of ether.[8]

With preliminary medication, the brain is better protected than if inhalation is used alone, and the reflex inhibition of the heart is impossible. This statement is proved with every ether anesthesia given. With proper preliminary medication, the patient goes to sleep quietly as in his own bed. During operation the pulse, respiration and blood pressure are normal. After operation, the patient usually awakens without nausea, vomiting, wound, or gas pains, and rarely requires a sedative. Without proper preliminary medication, quite often physical restraint of some kind is necessary if ether is used. When holding straps are used, a shudder, or tremor, such as is seen at electrocutions, passes over the body. During operation, response by pulse, respiration and blood pressure show that the brain is not as completely blocked as when preliminary medication is used. After operation, pain is the first reflex, and morphin is then given, which usually results in gas pains and other inconvenient reflexes.

When for any reason preliminary medication is omitted, ethylene blocks the brain more effectually than does nitrous oxid. During the war, morbidity and mortality were unquestionably reduced for thoracic patients by the method outlined, i. e., each patient receiving

8. Gwathmey, J. T.: New York M. J. **104**:825 (Oct. 28); 895 (Nov. 4) 1916.

three-eighths grain (0.024 gm.) of morphin, and nitrous oxid and oxygen (given to unconsciousness only), under positive pressure, thus completing the analgesia and anesthesia.

MAGNESIUM SULPHATE AND MORPHIN

The only change that I would now suggest would be to give the morphin in 2 c.c. of a 50 per cent. chemically pure sterile solution of magnesium sulphate instead of sterile water, each patient getting three-eighths grain (0.024 gm.) of morphin and 6 c.c. of a 50 per cent. solution of magnesium sulphate. Then this should be supplemented by giving ethylene, nitrous oxid and oxygen, and by omitting the supplementary ether except in unusual cases. Fifteen grains (0.97 gm.) of the salts of magnesium sulphate with morphin has been used many times as a preliminary to general surgery. We are, therefore, perfectly safe in using 6 c.c. of a 50 per cent. solution (3 gm.) as outlined above.

THE AMERICAN MEDICAL ASSOCIATION COMMITTEE

The American Medical Association Committee appointed to investigate "the toxic effects of local anesthetics," states that "morphin has a distinct field of usefulness in allaying the apprehension of nervous patients before operation, but it is necessary to sound a note of warning against the use of too large doses of morphin or the use of repeated small doses."[9] The opposite view is taken by Braun in his sixth revised edition; he states that "it is a mistake to torture a patient with fearful apprehensions, but in a majority of cases fear may be overcome by the preliminary use of scopolamin and morphin." He advocates enough morphin and scopolamin to put the patient to sleep, and if the patient is not asleep at the end of an hour, the dose is repeated. "The eyes are bandaged and the ears plugged with greased cotton. As a rule, the patient after operation does not know what has been going on."

PRELIMINARY MEDICATION AND BLOOD PRESSURE

An ordinary dose of morphin, one-eighth, one-sixth or one-fourth grain (0.008, 0.010 or 0.016 gm.) does not completely remove psychic impulses.[10] At the Woman's Hospital in New York City, with patients who have received a preliminary dose of morphin, a rise of blood pressure of "from 10 to 40 mm. systolic ordinarily, and, occasionally, on up to 80 and 90 mm. occurs just prior to the administration of the anesthetic."

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9. Mayer, Emil: Toxic Effects Following Use of Local Anesthetics, J. A. M. A. **82**:876-885 (March 15) 1924.

10. Coburn, R. C.: Blood Pressure in Operative Surgery and General Anesthesia, J. A. M. A. **82**:1748-1750 (May 31) 1924.

DIFFERENT METHODS

A preliminary of three-eighths grain (0.024 gm.) of morphin and 6 c.c. of a 25 per cent. solution of magnesium sulphate has been used by Lathrop [11] more than 400 times, and, in addition, from 1 to 1½ ounces (30 to 45 c.c.) of ether mixed with 4 drams (15 c.c.) of sweet oil, by rectum. Lathrop had previously used ether-oil colonically over 2,000 times with no untoward effect, but he prefers the technic just outlined. As magnesium sulphate is definitely synergistic with ether, and as the dose mentioned is so small that toxicity need not be considered, I prefer the 50 per cent. solution, and also to increase the ether to 2½ ounces (74.2 c.c.) with the addition of paraldehyd, from 2 to 4 drams (7.5 to 15 c.c.), and oil, 2 ounces (59.2 c.c.), all given by rectum. Paraldehyd adds nothing to the analgesia; it simply insures a perfectly quiet and safe sleep. With this preliminary (i. e., the three hypodermic injections and rectal installation), certain operations can be done without any additional local or inhalation anesthestic, although the majority of operations usually require either one or the other. It is our belief that over 80 per cent. of all hospitals and individual surgeons in the United States use some form of preliminary medication. It is also our belief, founded on the fact of the tremendous rise of blood pressure, previously stated, that most patients are better protected from psychic impulses if they are in such condition as not to realize what is taking place. When from the nature of the operation loss of blood and shock are apparently unavoidable, the loss of blood can be anticipated by giving 1 quart (178 c.c.) of tap water with 5 per cent. glucose, by rectal drip, following Well's technic,[12] starting two hours before the operation.

Psychic shock can be prevented by the employment of the three hypodermics and the rectal instillation, as just outlined. Ethylene, nitrous oxid and oxygen, given to unconsciousness only, with slight positive pressure (from 1 to 3 mm. of mercury), maintained when indicated, insures a complete brain block during the operation and a convalescence in which pulse, temperature, blood pressure and respiration, together with freedom from pain impulses, indicate a nearer approach to the ideal than has yet been reached.

40 East Forty-First Street.

DISCUSSION

DR. J. S. LUNDY, Rochester, Minn.: Last November, I began to use a machine by which it was possible to give nitrous oxid, ethylene and oxygen at one time, and to measure the flow of each. At that time, I believed there were certain advantages, and I still think so. Recently, here in the Mayo Clinic, in the obstetric department, we have used this combination of ethylene, nitrous

11. Lathrop: Personal communication to the author.
12. Wells, C. J.: Am. J. Surg. (Anesthesia Supp.) **37**:68 (July) 1923.

oxid and oxygen, with ether if necessary, during labor. The pains of labor are comparable to those caused by thoracic surgery, and therefore our experience with the various anesthetics in obstetric cases may be of interest.

As the cervix is dilating, I give nitrous oxid with a great deal of oxygen, aiming to keep the patient asleep, and to keep the color of the skin and blood pink, always. As soon as voluntary effort begins to reinforce the contractions of the uterus, I add a small amount of ethylene to the nitrous oxid and oxygen, and then increase the quantity of oxygen. As expulsion begins, I change from nitrous oxid to ethylene-oxygen, and am able then to control the pains without any cyanosis. As the head is delivered through the perineum, I use ethylene and oxygen only, with about 10 or 15 per cent. oxygen, or occasionally add a dram or two (3.7 or 7.5 c.c.) of ether.

The advantage is that if the head comes down against the perineum, causing too much pressure, and the obstetrician says, "Stop the pain," it can be stopped abruptly with ethylene and oxygen, thereby avoiding a good deal of laceration, and without letting the patient wake up. Also, one can change over to nitrous oxid and oxygen, and the pains will come on again without the patient waking up. Thus, by keeping the patient below the level of consciousness, we seem to control the pains on the principle of the blending of gases, which I think is as fundamental in anesthesia, as is the blending of metals in metallurgy, of notes in music, or of pigments in painting.

Dr. John L. Yates, Milwaukee: During the preliminary periods of experimentation at the United States Army Laboratory of Surgical Research, at Dijon, and, finally, in actual work in field hospitals in France, I was subjected very directly to the rulings of Dr. Gwathmey, and profited tremendously thereby. Dr. Gwathmey, himself such a master in the administration of anesthetics, fails to take into consideration that the rest of the world is less competent and therefore liable to err. The only things to be said of his work here presented are appreciation and gratitude.

Two points in the administration of anesthesia by positive differential pressure methods for intrathoracic surgery are noteworthy, and are based on observations made on animals and human beings. When positve pressure is employed in the administration of an anesthetic, pulmonary inflation is correspondingly increased, and with increase of inflation a greater amount of air containing various concentrations of anesthetics is exposed through a correspondingly increased alveolar wall to correspondingly larger amounts of blood in the pulmonary capillaries. The unwary may induce their anesthesia too rapidly by employing either too great concentrations of the anesthetics or too great pressures. A fatality is easily and promptly produced. This is not because the pressures favor gaseous interchanges but because the positive pressure, causing inflation, assures a more rapid interchange of gases and more intensive effects.

Dr. Gwathmey will recall that, in the first surgical experiments on dogs, there was a very high mortality rate from the anesthetic; a number of these animals died on the table. We were at first unable to explain their deaths.

There is a limit to the amount of positive pressure to employ even on a strong man, and it is apparently approximately 15 mm. of mercury. There is a temptation to exceed this limit or pressure when trying to blow up a deflated lung. When the limit is exceeded, the resultant type of pathologic emphysema causes such a distention of the air cells and excess pressure on the interalveolar capillaries that the back pressure can produce immediate acute dilatation of the right side of the heart. This is to be recognized as

a grave danger. We find in our records that, in our earlier work with the wounded, in a laudable desire to reinflate injured lungs to avoid excision, we did overstrain cardiac competence, and, in a few instances, induced a fatal, acute cardiac dilatation.

Dr. Charles G. Heyd, New York: A number of years ago, we published a paper on rectal anesthesia and after about seventy-five cases we discontinued it on account of a change of hospital service, but in the last eight months, we have been using the analgesia, particularly in operations around the neck and chest, and it is a surprising thing to be able to remove an adenocarcinoma of the thyroid with the patient talking to one, and not feeling any pain unless one makes unusual or violent traction. The absence of any cyanosis or flushing of the face and the absence of increased salivation are so marked that it is really surprising.

We have been in the habit of using morphin in larger doses than Dr. Gwathmey has mentioned, usually a sixth or a quarter grain (0.01 or 0.016 gm.), and not the serial gradation of an eighth grain (0.008 gm.). We have rarely combined it with magnesium sulphate, and there are two reasons for that: first is the apparent indifference of the nursing force to precision as to time of giving narcotics; second is the delay that sometimes occurs in a large hospital in obtaining the operating room at exactly the precise time that one indicates. In the New York Postgraduate Hospital, we have to give way at various periods to succeeding operators, so we are under the difficulty, in many cases, of being delayed in our entrance into the operating room. But the advantage, to my mind, of the serial dosage in the preliminaries is very great. Occasionally, we find a person that is unusually susceptible to morphin, and we have had one or two cases in which, so far as we know, the patients received only a sixth (0.01 gm.) of morphin and were completely under the influence of morphin for from six to eight hours.

I think that Dr. Lundy put it very well, that anesthesia is a matter of mixing. It is like the question that was once asked an artist, how he mixed his pigments, and he said with brains, and that Dr. Gwathmey has contributed today.

Dr. Frank C. Mann, Rochester, Minn.: Dr. Gwathmey's paper proved one thing to me very clearly; namely, that what we had anticipated is not true, that ethylene is the ideal anesthetic. I have been led to believe that probably here we did have the ideal anesthetic, but whenever we find some one who is skilled in the use of anesthesia and he starts combining a group of things with any other anesthetic, we know that we still have to find the ideal anesthetic, because when we get the ideal anesthetic it will not be one that is in combination with anything else. The future research that deals with anesthesia is going to deal with a development of one specific thing, and not with the combination of them, although I wish to emphasize that, at the present time, with the drugs we have, some having this action and some having that, some of the actions undesirable, some in which one wishes to increase a certain part of the action, as Dr. Gwathmey's method has developed, it is necessary to use these methods which he has presented; but the development of research will be along the line of production of one substance, because one can look back or one can look over the field of surgery in the United States—county hospitals springing up all over the country—and one cannot get enough anesthetists who will use the combination accurately that Dr. Gwathmey has presented.

Preliminary medication in anesthesia is not to be the final solution of the anesthetic problem, and while I wish to emphasize the great advantage of the methods that Dr. Gwathmey has presented in the skilful administration of anesthesia in the place in which it can be done and by the skilful anesthetist, for the majority of surgical operations we are going to have to depend on the use of one or, at the most, two or three drugs administered by some one who is not skilled or never will be skilled in the proper combination of the large number that we have.

As I say, the main thing I have gotten out of Dr. Gwathmey's paper is that we still have to search for the ideal anesthetic, that ethylene is not that.

Dr. Gwathmey has shown that he can use these different combinations to great advantage, and I know enough about the action of them to know that he is using them to get the most out of them for the purpose that he intends.

Dr. Edward S. Welles, Saranac Lake, N. Y.: I should like to ask Dr. Gwathmey if he is using magnesium sulphate in combination with morphin exclusively in place of scopolamin, or does he still use scopolamin and morphin occasionally? And he makes no mention of atropin. Does he leave that out altogether in preliminary medication, when it is a case that is going to require nitrous oxid or ethylene, after the local anesthesia, or without any local anesthesia?

Dr. T. C. Davison, Atlanta, Ga.: I should like to ask Dr. Gwathmey to give the proportions that he would recommend.

Dr. Carl A. Hedblom, Rochester, Minn.: I should like to ask Dr. Gwathmey what his observations have been regarding the effect of the combined anesthesia, that is, the morphin and magnesium sulphate, on the coughing reflexes. I have always felt that caution was necessary in giving morphin in cases of pulmonary suppuration, in cases in which there is a great deal of sputum; if one gives enough morphin to obliterate the coughing reflexes or to reduce the irritability of the bronchial tract, the sputum may be retained until the bronchial tree practically fills up, and then if the patient is in a rather weakened condition, the shift of position or something else brings on the cough, and he is apt to spill the pus into other portions of the bronchial tree. I have seen some very serious consequences of just that sequence.

I think that we, as a group, are especially interested in anesthesia in thoracic surgery, and it seems to me that, in cases of pulmonary suppuration, the matter of retention of sputum has to be given very careful consideration in relation to the preliminary medication used.

Dr. James T. Gwathmey, New York: I should mention that Lundy deserves very great credit for designing an apparatus in which you can use nitrous oxid, ethylene, carbon dioxid and oxygen separately or combined, or in sequence, as indicated.

According to all observers, ethylene reduces the blood pressure and respiration. The respiration should be reduced in thoracic surgery. On the other hand, nitrous oxid stimulates both the respiration and the circulation to a certain extent. Now, by juggling these two gases one against the other, one can keep the respiration just where one wants it with very little trouble. For instance, if the reflexes are abolished to a point at which one thinks there is a slight danger, one can switch off ethylene and give nitrous oxid to keep the patient under anesthesia, and the reflexes very gradually come back; if now the patient gets a little active so that one expects some unpleasantness, such as nausea or vomiting during operation, one should add a little ethylene, and the patient sinks back again.

Dr. Mann missed the idea of my paper entirely. One does not require an expert anesthetist when one gives preliminary medication so much as when this is withheld. If one looks at the problem of preliminary medication as just presented, it means the abolition of third stage inhalation anesthesia. With preliminary medication, one does away in a very great measure with the necessity for an expert anesthetist.

With the preliminary medication that I suggest, the three hypodermic injections of one-eighth grain (0.008 gm.) of morphin and 2 c.c. of magnesium sulphate, 50 per cent. solution, one never gets a patient anesthetized so completely that he has a relaxed tongue, and, therefore, one does not need an expert anesthetist. Now, if one adds to that medication procain for the skin and peritoneum, one requires no further anesthetic, if the person is skilful in the use of local anesthesia. On the other hand, the efficiency of any hospital, especially a large clinic, is determined by the time that the surgeon can use in surgery; therefore, instead of procain for skin and peritoneum, inhalation anesthesia may be used.

I know that Dr. Mann's idea about a single agent is the idea that is prevalent, and the subject of synergism is usually repulsive to physiologic workers. All are trying to get some one agent that one can use as a man would use a rifle. Very little is known about synergism today, but Meltzer stated exactly what synergism was without calling it by name when he made this statement: that if one uses a small amount of ether, insufficient in itself to produce anesthesia, and a small amount of magnesium sulphate, insufficient in itself to produce anesthesia, and combines the two results, one gets a deep anesthesia. Now that is a definition of synergism. It is a case of two and two making six or eight instead of two and two making four, but it has never been proved until recently, when it was proved by Dr. Chas. W. Hooper (personal communication).

Personally, I don't think that we will ever get a single agent that is ideal, but by supplementing this preliminary medication with either inhalation anesthesia or procain for the skin and peritoneum, we have something that is more nearly ideal than anything in use at the present time.

During the war, our men would go from the operating table in a condition to receive fluid by mouth; they were perfectly conscious and yet they were analgesic, in an entirely different condition from the same class of patients who had inhalation ether, and who took from two to four hours or more to get over the effects; this condition certainly doesn't add to the safety and comfort of any patient.

Scopolamin may add directly to the respiratory arrest of a patient even in small doses, but one is safe in using magnesium sulphate in the dosage given. There is no objection to using atropin.

In regard to the proportions of the gases, two parts of oxygen should be given all through the operation; at first, four parts of ethylene and two parts of nitrous oxid should be given and this should be reversed or the amounts modified according to the condition of the patient.

In regard to the cough reflex, this can be retained by modifying the inhalation anesthesia.

I have already mentioned the one danger in giving preliminary medication, and that is overlapping; if one attempts to use the same degree of anesthesia with preliminary medication as without it, the patient is immediately thrown into some degree of shock.

CLINIC ON ABSCESS OF LUNG

Saturday, June 7

WILLIS S. LEMON, M.D.

A woman, aged 37, had been perfectly well until eighteen months before, when she began to feel poorly, losing weight and color, fatiguing easily, and coughing. Pregnancy occurred, and immediately she became better. She went through with her pregnancy normally until within the last month when symptoms recurred. A tumor in the chest was diagnosed. The cough became more irritating, persistent and paroxysmal in character. It was not productive of any great amount of sputum, and, contrary to the usual experience, the patient did not expectorate blood. The cases of hemorrhage, as seen at the clinic, are classified approximately as follows: (1) tuberculous; (2) chronic heart disease; (3) abscess of the lung, and (4) bronchiectasis and allied affections, such as pneumonoconiosis, which usually produces bronchiectasis. Aneurysms and tumors and miscellaneous growths of various kinds within the lung, whether primary or secondary, are productive of hemorrhage which appears either as streaks in the sputum, or as actual free bleeding.

This woman did not have hemoptysis at any time. One could see that she was thin, having lost from 15 to 20 pounds (6.8 to 9 kg.) in weight, which was a great deal, considering that her normal weight was only 125 pounds (56.7 kg.). She was also anemic, having a blood picture of only about 60 per cent. both in color and in count, and she tired easily.

She likened her cough to that of whooping cough, without the whoop. Such a cough affords information concerning localization. The patient had noticed that, if she changed her position from a sitting to a prone position, she coughed; if she changed from horizontal to vertical, she coughed. Cold air also produced cough.

The chief factors, then, in her case were: (1) eighteen months illness; (2) an insidious onset; (3) cough and loss of weight, and some sputum; (4) a lapse during pregnancy and recurrence following pregnancy; (5) the finding of a tumor; (6) symptoms which generally accompany growth of the hilum (hers was a hilum growth; one could foresee that without the use of roentgen ray, of course), and (7) the presence of metastatic lymph nodes, diagnostic evidence frequently overlooked.

If tumor of the lung metastasizes, it usually does so to the neighboring lymphatic nodes. On examination of a patient with signs and symptoms suggesting malignancy, it is important to palpate carefully the supraclavicular areas. Frequently, a hard mass will be found in the right supraclavicular space; this is much more suggestive than when it is on the left side because it may be due to metastasis from below the diaphragm; on the right, it is usually due to metastasis above the diaphragm.

This node can be felt by gentle pressure of the fingers; force should never be used since the lymphatic gland can be detected much easier by gentle than by forced pressure.

In the roentgenogram, one could see the infiltration passing into the right lung from the hilum area. This picture in itself was not sufficient evidence to warrant a positive diagnosis of malignancy. If there were in the parenchyma a single metastatic area, the picture would be classical of primary carcinoma of the lung. Other malignant tumors must be considered, including lympho-

sarcoma and dermoid. As most of the latter patients appear between the ages of 16 and 30, one naturally thinks of the possibility of a dermoid that has become malignant. I do not believe that a positive diagnosis can be made without removing the metastatic lymph node and examining it pathologically. The patient had improved under radium treatment. I doubt if future improvement would be as rapid and spectacular under radiant treatment.

This patient had been under my care for several years. She had a lung abscess which followed tonsillectomy, done elsewhere in 1916. Ether anesthesia was used, and even though the cough reflex may be suppressed by the anesthetic, the pharyngeal reflex may be retained. Mucus and blood are insufficient irritation to produce cough. Respiration may then take place.

The sequence in this case was as follows: pneumonitis occurred; necrosis followed, and shortly afterwards, an expulsion of a large quantity of pus. Hemoptysis appeared early, lasting, with the filling and emptying of pockets, four years. In August, 1922, empyema developed. Empyema may develop in any of these cases because of the direct connection between the deep and superficial lymphatic vessels; infections within the lung are easily conveyed to the pleura from the parenchyma. The abscess which lies peripherally may rupture into the pleura and produce empyema. In this case, empyema followed and a rib resection was performed, the drainage sinus remaining, until January, 1921, when another rib resection was performed to facilitate further drainage. The extrapleural thoracoplasty was performed because several years had elapsed between the preliminary infection and the date of examination in the clinic, so that the whole picture had changed from that of an abscess to one of many complications. If necrosis and abscess appear within the chest and necrosis is succeeded by fibrosis, then bronchiectasis must be the end-result. Thus, in many instances, there is a combination of abscess being succeeded by bronchiectasis, which, extending into the periphery of the lung, may produce abscess, as happened to this patient. She came back to me a year ago with symptoms almost identical with her early symptoms; a cough, producing foul expectoration, signs of definite cavitation in the right axilla, malaise, fever, general weakness and anemia.

The thoracoplasty had reduced the size of the diseased side greatly, but it had not removed the important lesion. The source of infection was still in the chest; the patient might, of course, break down under acute infection. For this reason, one always stresses the importance of living so as to avoid acute infections. They always cause a recurrence of symptoms and are very likely, by extension or aspiration, to invade other portions of the lung.

The patient was now as well as she had been for years, not as well as she was, naturally, before she had this disease.

The next case was very interesting. The patient normally weighed 162 pounds (73.5 kg.); when I first saw him he weighed 115 pounds (52.1 kg.). His trouble started in 1917 with a cold. It developed into a cough productive of very foul sputum almost at its onset, and six months later a severe hemorrhage of about 8 ounces (236 c.c.) of pure blood occurred. Later, he had hemorrhages frequently. These were controlled by means of artificial pneumothorax. The lung was in collapse for six weeks. He was operated on here for the first time, in 1918. His breath and his expectoration were so excessively foul that boarding houses and hotels would not admit him, and he was sent immediately to the hospital and kept in a ward away from the other patients. He had a cavity in the upper lobe of the lung, and it was difficult to decide whether or not it was tuberculous. With an insidious onset, a preliminary hemorrhage

and an apical abscess, the provisional diagnosis, naturally, was tuberculosis, and yet after the most careful examinations and after repeated sputum analyses, we were unable to find tuberculosis bacilli in the sputum.

Dr. Hedblom operated, opening into and draining the abscess. The patient had an extremely stormy convalescence; finally, however, he began to improve. He went home, and, a year later, returned prepared to submit to a second operation for closure of a bronchial fistula which existed at that time and which still exists. At this examination, there was evidence suggestive of tuberculosis in both apexes; this was confirmed by roentgen-ray examination of the chest and finally by finding tuberculosis bacilli in the sputum. The patient was placed on the usual tuberculous regimen; he gained weight, strength and health, and one could see him now practically a healthy man, with the only drawback that he could not take a full bath, because he had a fistula in the chest. There was a small amount of discharge from the sinus, but essentially the man was healthy.

This case illustrates very well Dr. Willy Meyer's work on lip fistula in chronic cases; although the fistula is not an addition to beauty, neither is it a matter to be particularly concerned about. This man has lived since 1918 with this opening in his chest, and I doubt very much if Dr. Hedblom will consider stirring it up. In the roentgenogram, all that could be seen was a shadow due to fibrosis of the lung and pleura which does not allow light to penetrate. It is a fact that abscesses may be insidious in onset, apical in location and nontuberculous in origin. Yet, if they are insidious, if they are introduced with hemorrhage, if they are apical, then they must be considered tuberculous until they are proved otherwise.

Another patient, who was likewise a husky man, in very good health, had a history much the same as that in the second case. He had an operation on the throat under ether anesthesia, and developed a lung abscess, bronchiogenic in character, and empyema, complicated by a bronchial fistula. He now had a fibrosed lung. His general health was good; his weight had kept up to normal. There were still some small fistulas present, in spite of repeated attempts to close them by the cautery, and recently by the silver nitrate method. They were very small now, and caused no trouble, but possibly some other means of closing them may be found.

Another patient had a rather unusual history. At one time, he was cleaning out a hog pen when it was excessively dusty; he breathed a good deal of this material, and he said that for months he was able to feel a constriction in the chest. He had dated his disability from this time. With the actual onset of the trouble about seven years before admission, he had a very severe chill which was followed by vomiting. He was well, however, after this, except for a morning cough followed by a pain in his right side. Coughing spells were severe, nonproductive at first, and then finally productive of huge amounts of sputum. Later, the purulent material was mixed with blood. The patient had had four operations in attempts to drain the abscess and close it, before coming to us for his final operation and cure. This man has been working on his farm for a number of years. There was an area in the chest unsupported by ribs, which bulged when he coughed, but his chest functioned perfectly.

DISCUSSION

DR. CARL A. HEDBLOM: We occasionally have a patient with a lobe of the lung completely riddled with large bronchial fistulas. Here is such a case. This was originally, I think, a case of gangrene of the lung with secondary empyema.

This young lady happened to be in the hospital when Dr. Duval was visiting us, and I showed her to him. He said, "Doctor, it is perfectly useless to attempt anything in this case." That, coming from such an authority, caused me almost to lose hope, but I persisted. I had already resected the lower half of the scapula and several ribs for exposure. I then used the cautery. She had to return home and remained away until now. She came back at this time with the exposed area perhaps one-quarter the size it was then and with a large proportion of the fistulas closed. We shall continue with the cautery treatment, and I think we shall eventually secure complete healing. Her general condition, as you will note, is good.

Here is a second patient presenting a similar condition. He came with a history of a chronic abscess of two years' standing. I evacuated perhaps a half liter of gelatinous pus at the original operation. The cavity was the size of a large grapefruit, and the presenting surface of the lung was riddled with fistulas. He still has a large cavity with multiple fistulas. I have not advised an extensive plastic operation here because of the large amount of drainage from the bronchi. If we should succeed in closing the fistulas and he did not have any more serious complications, he would have to raise by mouth the pus that forms. He certainly is better off to have it drain through the wound. He is in good condition and works every day. He came 730 miles, driving his car himself all that distance in three days to give us the opportunity to see him.

Here is still another case. This patient had an unrecognized empyema draining into the lung. When I first operated, I counted thirty bronchial fistulas, and there were many more minute ones. We have performed a several stage plastic operation and have healed the fistulas with silver nitrate cautery. He has gained 23 pounds (10.4 kg.), and the fistulas are almost all closed. They will all heal shortly.

Dr. Lemon has shown three patients with pulmonary abscess with residual bronchial fistula illustrating different principles of treatment. In the case of this patient, no attempt has been made to close the fistula. It is an example of what Dr. Meyer had aptly named a "lip fistula." The fistula causes the patient no inconvenience except for a small dressing daily, and he is in splendid condition. The chief reason why we have not advised an operation for closure of the fistula is the possibility that a tuberculous process is still present in the lung.

The other patient had a very large fistula which caused him considerable annoyance. He said to me that when he attempted to swear at his mules he only whistled through the back of his coat. The preliminary operation for plastic closure involved resecting a very large, dense, bony ring. The opening was too large to attempt to suture. The skin flap closed it effectively.

The third patient, after a plastic operation, still has a small fistula. The reason for this is that a sufficiently extensive rib resection was not done to bring the fistula bearing surface on a plane with the skin used as a plastic. A deep sulcus also results. The residual fistula is closing following the use of silver nitrate cautery.

In most of the cases of bronchial fistula, particularly in cases with multiple small fistulas, we secure healing by persistent silver nitrate cautery. This destroys the epithelial outgrowth from the bronchus and results in granulation tissue formation, which, in turn, leads to scar tissue shrinkage. It is quite remarkable how effective this simple treatment is. We have had patients in whom the lung was so full of fistulas that it was like a sieve, and some of the fistulas up to a half centimeter or more in diameter entirely healed by

this method. The actual cautery tends to enlarge the sinus which defeats its own purpose. It is important not to attempt to close a bronchial fistula that is discharging any amount of pus. In one such case in my experience, a brain abscess resulted. In some cases, I have cauterized and then sutured the bronchus with good results.

CASE OF POSTTONSILLECTOMY PULMONARY ABSCESS

I am showing this patient, who is fortunately now entirely well after undergoing a serious operation for drainage of a posttonsillectomy pulmonary abscess, to emphasize the importance of prophylaxis against infection of the lung during tonsillecomy. We have had fifty-three cases of posttonsillectomy abscess at the Mayo Clinic; all except two have had tonsillectomy elsewhere. In my opinion, most posttonsillectomy abscesses are of the aspiration type. More than 90 per cent. of over 400 cases were done under general anesthesia.

It seems to me that it is time for an organization such as this to go on record as emphasizing the importance of prophylaxis against posttonsillectomy pulmonary abscess.

CASES OF BRONCHIECTASIS

F. W. Gaarde, M.D.

There is a certain unilateral type of bronchiectasis in which it is definitely worth while to attempt relief by surgery. When it is bilateral, there is usually not enough benefit obtained by collapsing one side to warrant surgical procedure. We have three unilateral cases here that have been greatly benefited by graded collapse, and I shall show three more later, which are almost completely cured by this method.

All these cases originated in a rather severe respiratory infection. The pathology is usually that of infection with injury to the walls of the smaller bronchi, which are dilated by the force of coughing. These dilatations eventually form permanent cavities because of the associated fibrosis. To put sequence of events in order, I think one would say, first, injury to the bronchial tube; second, dilatation by coughing; third, fibrosis, and, fourth, coincident with the fibrosis, permanent cavitation or bronchiectasis.

In Patient 1, the trouble began, in 1916, and it was a rather slowly progressive thing; apparently, it will sometimes take one, two or three years for this fibrosis, bronchiectatic process to complete itself. In 1918, he first noticed foul sputum, although his sputum had been gradually increasing in amount during the two years. He came here, in 1923, and Dr. Hedblom did a many stage collapse operation, which has reduced the amount of sputum from 16 to 8 ounces (473 to 236 c.c.). He has returned for further consideration, and, if his general condition and vital capacity reading so warrant, further collapse will be attempted.

The second patient has had a chronic cough and some sputum ever since he was an infant, and the original cause was the aspiration of a wheat beard. Nevertheless, he got along pretty well, until 1923, and at that time had a tonsillectomy. This was apparently followed by an abscess, and when he came he was having 24 ounces (710 c.c.) of sputum a day, and, by similar collapse, this has been reduced to 5 or 6 ounces (148 to 178 c.c.).

I wish to say a word about the vital capacity in these patients. The first patient shown has a vital capacity of 2,269 c.c., and we estimate his normal at 3,500 c.c. Therefore, when Dr. Hedblom has gone over him and decided

that further collapse is feasible, we feel that this can be done with caution, of course, without too seriously endangering him as to his vital capacity.

The second patient has a vital capacity of 2,720 c.c., with an estimated normal of 3,500 c.c. When one considers that he has only a slight reduction in his vital capacity and has reduced his sputum from 24 to 5 or 6 ounces (710 to 148 or 178 c.c.), I believe it will be agreed that further surgery is warranted in an attempt to reduce the amount of sputum still further.

The third patient was 19 years of age, and he was well until seven years ago. At that time, he was playing football and the ball hit him on the head; he had a brass upholstery tack in his mouth, and by a quick breath the tack was aspirated. He coughed and had a little fever for about two years, but it was two years before his cough became productive and foul, again showing the slow process of development sometimes seen in bronchiectasis. In 1922, he had a lung abscess drained, and he came here with a draining sinus, in 1923. The tack was found by roentgen-ray examination. The problem was how to remove this tack. It seemed a logical thing, since he had a sinus, to follow it up to the tack, and Dr. Hedblom did this and removed the tack through a 3 inch (7.6 cm.) incision. Tubercle bacilli were found in smears from the sinus, which probably accounts for the fact that it has not healed, and probably will influence Dr. Hedblom as to whether or not surgery is indicated. There has been no collapse done in this case, and we find his vital capacity reduced almost half. We have not taken vital capacity readings on all these men before they were operated on, but we have taken preliminary readings on a good many of our patients and very often find a reduction. Therefore, we feel certain that the collapse operation is not entirely responsible for the reduced capacity; in fact, in many cases, it has contributed only a minor part.

I shall not go into a detailed history of the next three patients, except to say that the process began in each of these girls with the general principles that we laid down for bronchiectasis, injury in the form of pneumonia in two, and, in this patient, measles with severe and prolonged bronchitis.

Each of these girls had in the neighborhood of 24 ounces (710 c.c.) of foul sputum, had reached a point where it was rather disagreeable for them to live, as they all said, and these two being under 20 years old and the third still a young woman, we are very glad to present the results obtained. Mrs. C. had 24 ounces (710 c.c.) of sputum, and has reduced that to none. When she catches cold, she may have an ounce or an ounce and a half (30 or 45 c.c.); this lasts a week or ten days and then disappears. Miss S. is not quite through; she still has some sputum, and is back for Dr. Hedblom's consideration of further collapse. Mrs. L.'s sputum is also reduced from 24 ounces (710 c.c.) to practically none, and she has very little cough.

Now, there is not very much to see except the same incisions that were seen before. It would be difficult to collect a group presenting more striking clubbed fingers than we see in these patients. It is remarkable that the lung could be collapsed, sections removed from the ribs, and the patient still maintain a pretty good spine.

DISCUSSION

DR. CARL A. HEDBLOM: May I say just a word on the matter of the amount of collapse? The question has been asked me several times, Why not limit oneself in these bronchiectasis cases to the technic that is used in the tuberculous, that is to say, resecting only the posterior portion of the ribs?

Now, here is a young lady on whom we did only a partial operation, the reason for which I shall not go into now, and if I should take time to show

the roentgenogram it would show a partial collapse only, and she still has a moderate amount of sputum, from 6 to 8 ounces (178 to 236 c.c.), and I think that is the answer, at least for this particular case, to the question, Is it necessary to do such a complete operation as to remove the whole length of the ribs?

It is my opinion that the success we have had with the extrapleural thoracoplasty has been due to the fact that we have done a very thorough job, and the reason we have been able to get away with a complete resection has been that we have been willing to do the operation bit by bit. It is more or less like cutting a dog's tail off by inches, but if you make it a painless procedure he doesn't mind and one gets results. The collapse by a one stage or two stage operation yields a very high mortality. We have had no mortality in the bronchiectasis cases.

DR. F. W. GAARDE: I haven't any more to say except that one must be sure that these cases are unilateral.. There could be a small degree of bronchiectasis without a definite positive roentgen-ray picture of bronchiectasis, which gives more of a bronchial thickening, and there one must depend largely on the physical findings, or on bronchoscopy.

I should like to say a word about the medical management. It doesn't offer the patients very much; when we get a bilateral case, we give the patient postural drainage night and morning and have him inhale increasing concentrations of beechwood creosote steam. It is a purely mechanical means of relieving the distress so that he coughs less during the day, and beechwood creosote will relieve part of the foul odor or disagreeable taste for the patient.

FOUR CASES

ARTHUR E. MAHLE, M.D.

CASE 1.—L. M., a woman, aged 24, with tuberculous osteomyelitis of the manubrium sterni, in 1921, developed some general malaise, indefinite pains in the shoulder and abdomen, but had no noticeable fever, night sweats or weight loss. A few months later, a small mass developed over the sternum. Her home physician excised this mass, which fluctuated, and obtained pus. He said that the infection did not involve the bone. She came to the Mayo Clinic one year later with a draining sinus over the manubrium sterni which had been present since the first operation.

Examination: Her general condition was fair. The hemoglobin was 72 per cent., and roentgen-ray examination of the chest was negative. Examination of the sinus discharge showed tuberculosis bacilli present. Dr. Hedblom explored the sinus and resected a portion of the manubrium sterni. Histologic evidence of tuberculosis was found in the tissue removed. The patient then left and was advised to go South, and, in addition to living out of doors, to have heliotherapy and to use the quartz lamp. The wound healed completely, about a year later, and has remained healed since.

CASE 2.—Dr. W. H. C., a man, aged 50, with a posttyphoid sinus of the right anterior chest wall, came to the clinic, in April 1924, complaining of a draining sinus of the right chest of one year's duration. Two years before, he had had typhoid fever, and at that time he was in bed for five months, passing successively through parotitis with suppuration; nephritis, high blood pressure, which was variable in character, being high one day and low the next; double optic neuritis, with central blindness and floating opacities, and a typhoid spine with marked hypertrophic changes, which had made necessary

the wearing of a brace, the use of crutches for a while, and now a corset. He had had severe pain in the right kidney area, much albumin in the urine, and it was thought that he had an abscess which had ruptured and drained into the urinary tract. Typhoid bacilli had been recovered from the parotid and chest wounds and the urinary tract. He had been very frail and weak, and was 50 or 60 pounds (22.7 or 27.2 kg.) under his normal weight. For the last eight months before coming to the clinic, he had been convalescing in the West; his general condition had improved, and, except for sciatica and inability to bend, he felt well enough to return to work.

Examination: He was a well developed man about 14 pounds (6.4 kg.) under normal weight. The lower dorsal and lumbar regions of the spine were rigid, and the patient felt considerable pain on reclining. There was a large sinus with moderate seropurulent drainage emptying into the anterior axillary line at the fourth interspace. A probe inserted in the sinus reached almost to the sternum. The specific gravity of the urine was 1.023; there was a trace of albumin, a few red cells and pus cells. Typhoid bacilli were found in the smear made from the discharge from the sinus. Roentgen-ray examination of the chest, kidneys, ureters and bladder was negative. Roentgen-ray examination of the vertebrae revealed arthritis of the second, third and fourth lumbar vertebrae.

April 25, operation was performed by Dr. Hedblom under local and nerve trunk anesthesia of the third, fourth and fifth nerves. An incision was made in the fourth interspace extending from the anterior axillary line almost to the median line anteriorly and the fourth intercostal space. About 12 or 14 cm. of the fifth rib was resected, including the proximal portion and the cartilage. There was a great deal of scar tissue, which made local infiltration unusually difficult. The sinus extended nearly to the median line and involved the cartilage of the fourth rib. The fourth rib, therefore, was resected at about the same level as the fifth, and the intervening inflammatory tissue was removed in toto. After the fifth rib had been resected, a slight tear was seen in the pleura. This was promptly closed by pressure and sutured. The cartilages of the resected ribs were thoroughly removed. At the end of the operation, there was no evidence of a sinus.

The immediate postoperative condition of the patient was good. On the fifth day, his temperature rose to 100, and his pulse rate was also 100. On the sixth day, the temperature dropped to normal and remained so throughout convalescence. He was out of bed on the eighth day, and was dismissed from the hospital eight days later. When he left the clinic, June 15, the wound was practically healed, and his general condition was excellent.

Case 3.—K. T. G., a man, aged 33, with fibroma of the right side of the chest wall, ten years before admission, noted a small mass over the right sternum. It grew to its size in two or three months. He first was admitted to the clinic eight years before this admission, at which time there was a hard immovable mass, approximately 6 cm. in diameter, at the costosternal junction of the right sixth and seventh ribs. Roentgen-ray examination of the chest was negative. At that time, apparently a portion of the tumor was removed, and the cartilage of the fourth and fifth ribs resected by Dr. Robinson. The patient returned, three years later, with a mass below the previous operative scar which was hard and immovable. Dr. Hedblom, in a two stage operation, exposed and resected the tumor. It was found that the tumor lay underneath the sternum at about the level of the fourth rib and was adherent to the pericardium. Pathologic diagnosis, at this time, was fibroma. He has

returned to us several times since, and at no time have we been able to demonstrate a recurrence.

CASE 4.—G. H., a boy, aged 3 years 9 months, with right empyema, was admitted to the Colonial Hospital with temperature, 104, and pulse, 160. Roentgen-ray examination showed the right chest filled with fluid. Aspiration of the right chest yielded frank pus, and a tube was inserted through trocar and cannula, and irrigations with salt solution were carried out. The temperature gradually dropped, the general condition improved, and, two weeks later, a simple rib resection was performed. The wound promptly healed, and has remained closed since. At the present time, a year and a half later, he has a good respiratory excursion of the right chest and roentgen-ray examination is negative. In every way, he appears as a normal child.

DEMONSTRATION OF A RECURRENCE OF EMPYEMA AND A DIAPHRAGMATIC HERNIA

CARL A. HEDBLOM, M.D.

This patient, aged 64, in 1903 developed acute pain at the right costal margin and later symptoms pointing to a pleural effusion. He was thought to have an acute cholecystitis with an abscess perforating the pleural cavity. He was operated on by J. B. Murphy who did two drainage operations. Drainage persisted for sixteen months, when the sinuses closed. He held a responsible position and remained entirely well for nineteen years, until 1922, when, after a severe cold, he developed pain in the right side and shoulder with some fever and malaise. He then improved. After five months, a lump appeared in the right axilla which ruptured spontaneously at the site of the old drainage. He presented himself for treatment at the Mayo Clinic, and this sinus was found to lead to a cavity occupying the upper lateral aspect of the pleural cavity to the dome.

In a recent series of more than 400 chronic empyemas, I have found three distinct clinical groups: First, those in which one or more drainage operations had been done and in which the drainage had persisted continuously, comprising 50 per cent. of the cases; second, those unrecognized as empyema patients and so never drained, constituting about 23 per cent.; third, those who had had drainage but in whom the drainage opening healed over and remained healed for a period of from a few weeks to many years, about 27 per cent. The latter group, I found, consisted of two subgroups: One contained the great majority of cases in which the empyema cavity probably had persisted throughout. One patient, for instance, for a period of fourteen years after the drainage opening healed was able to obtain a splash inside the chest at will by shaking himself. The other smaller subgroup included patients who were perfectly well and at work for long periods of time. As I have mentioned before, I have observed one patient during the period of formation of the recurrent empyema.

This patient had remained entirely well and held a responsible position for a period of twenty years before the recurrence. I regard him as an example of a true recurrence of empyema at the site of the former cavity.

Here we were confronted with the problem of how best to deal with a chronic empyema in a man past his prime in years though not in spirits or capacity. Rather than do an extensive plastic operation, which would include resection of the upper ribs, we decided on a conservative type of treatment using surgical solution of chlorinated soda (Dakin's solution) irrigation and

supplementing this treatment with a minor plastic operation. His cavity is entirely healed and he is in splendid condition.

This patient had a traumatic diaphragmatic hernia resulting from a bad crush in a railroad wreck. Following recovery from this accident, he developed recurrent attacks of abdominal pain, colicky in character and so severe that he writhed with pain. It was thought that he had gallstone colic. He had had frequent attacks for two years before he came here. Dr. Balfour operated on him, suturing the rent in the diaphragm. He has been entirely well since.

Many patients with diaphragmatic hernia give somewhat similar histories. The last patient we had, just a few weeks ago, had been operated on for appendicitis, for gallstones, and for duodenal ulcer without the hernia being discovered. We made the diagnosis on the roentgen-ray examination findings. At operation, he was found to have a gastric ulcer and also a large diaphragmatic hernia. The association of gastric ulcer and diaphragmatic hernia is not so uncommon. In any patient who develops abdominal or thoracic symptoms following severe trauma, a knife stab, or a bullet wound in the lower portion of the thorax, diaphragmatic hernia should always be thought of. There also are, of course, the acquired and congenital types which may develop similar symptoms.

Let me mention that chondroma is the most common chest wall tumor aside from fibroma. It is usually considered benign, and is allowed to go untreated more or less indefinitely. In many cases, the tumors are about 20 cm. in diameter; they develop into sarcoma, recur and kill; so I consider chondromas at least potentially malignant, and believe they should be treated as such. Early removal can be done with relative safety.

CARDIOSPASM

PORTER P. VINSON, M.D.

I wish to say a few words in reference to cardiospasm, and point out several of the diagnostic features of this condition as differentiated from carcinoma. Of course, carcinoma of the cardia is the lesion that most often simulates cardiospasm. Benign stricture at the cardia is very rare; in fact, I have seen only one case.

The diagnosis of cancer can be made on three or four points without any endoscopic examination or roentgen-ray examinations. If a man more than 40 years of age has had dysphagia for a year or less, without a history of previous trauma, and his dysphagia has been progressive, beginning with solids, then semisolids and, eventually, liquids, the diagnosis of cancer is 90 per cent. correct.

It is rather necessary to know the amount of dilatation of the esophagus above the point of spasm before these obstructions are dilated, because if there is wide dilatation of the esophagus above the point of spasm, a higher pressure may be used with greater safety than if there is slight or moderate dilatation. Ordinarily, patients with cardiospasm can be cured by one stretching, and 75 per cent. of them remain well. Occasionally, it is necessary to dilate again, and most patients can be permanently cured if the stretchings are repeated.

The first patient illustrates a group of cases that have, I believe, been wrongly classified as cardiospasm. This man has a long history of intra-abdominal discomfort with colic strikingly like gallstone colic, probably due to disease in the gallbladder. He has had jaundice, and, in the last two or three years, has begun to have dysphagia. At the present time, the roentgen-ray

examination of the esophagus is entirely negative; there was no evidence of obstruction in the esophagus to the passage of a sound, and yet this instrumentation has given him complete relief from dysphagia and also complete relief from the epigastric distress that he had had after meals for a number of years.

Cardiospasm will not be cured by the passage of sounds. In this case, the result may not be permanent, and it may be advisable, later, to use a hydrostatic dilator, stretching the cardia moderately, in order to try out the effect. This patient's condition should be called spasm at the cardia, as differentiated from cardiospasm, in which there is a definite obstruction of the barium meal at the cardia.

THORACIC OPERATIVE CLINIC

Saturday, June 7

CASE OF BRONCHIECTASIS

A. E. MAHLE, M.D.

CASE 1.—A man, aged 23, with a clinical diagnosis of bronchiectasis of the right lower lobe, three years before admission, had developed a bronchitis with an associated pleurisy, from which he apparently recovered. One year later, he developed "influenza," and since that time had had a persistent cough and had raised a greenish yellow sputum, varying in amount from 2 to 6 ounces (59 to 178 c.c.) a day. One year before admission, he had three attacks of slight hemoptysis and, in the last six months, had lost 20 pounds (9 kg.) in weight. He had never had night sweats, but occasionally had some afternoon temperature. Specimens of sputum examined elsewhere had always been negative for tuberculosis bacilli.

Examination: Physical examination of the chest was practically negative. The roentgen-ray examination showed some infiltration of the lower right lobe (bronchiectasis). Examination of the sputum was negative for tuberculosis bacilli and elastic tissue. Hemoglobin was 70 per cent. Bronchoscopic examination showed a unilateral bronchiectasis with a dilatation of the right bronchus to the lower lobe. The patient was then given a complete artificial pneumothorax collapse of the right lung, and his sputum decreased to less than an ounce (30 c.c.) in twenty-four hours. It was then decided to do a graded extrapleural thoracoplasty for permanent collapse.

DISCUSSION

DR. CARL A. HEDBLOM: This was a case of unilateral bronchiectasis. We were uncertain, after the examinations were completed, as to whether or not it was unilateral, and so we did a pneumothorax collapse as a therapeutic test. The sputum decreased to an ounce (30 c.c.) or less. We concluded, therefore, that we were dealing with a case of unilateral involvement and so one suitable for extrapleural collapse of the chest wall.

We began with a paravertebral incision under combined local and ethylene anesthesia. I cut the periosteum at the upper and lower margin of the rib, leaving the portion covering the outer aspect of the rib attached and removing it with the rib. This will, I think, in part, prevent regeneration of the rib. It is not feasible to try to remove all the periosteum in a case such as this in which the parietal pleura is not thickened. That would mean that one would inevitably break through into the pleural cavity, which we must avoid doing on account of the danger of empyema in case the wound becomes infected.

I call attention to this curved periosteal elevator which I had made, and which is now on the market. It enables one to keep close to the rib, between it and the periosteum one is separating.

We cut the ribs proximally very close to their articulation with the transverse process; then by a little traction we could get an additional centimeter or two distally, taking out in all about 10 cm. Then we identified the intercostal nerve. The intercostal vein serves as a landmark to it. We injected procain as near the spine as possible on the theory that it would block the tendency for

the alcohol to produce pain at a point along the nerve where it was so slight in amount that it failed to anesthetize. We injected alcohol a little distally, just enough to produce an edema of the nerve. The alcohol injection, I regard as almost essential to the operation. It serves a threefold purpose. In the first place, it makes the convalescence relatively painless. This is very important, in view of the fact that a five or six stage operation is to be done. If the convalescence is painful, the patient is apt to lose his morale and refuse further operation. So far I have never had the slightest difficulty in that respect. Second, cough is relatively painless if the intercostal nerves in the operative field are anesthetized. This is a great safeguard. These patients, by virtue of the tolerance of their bronchial tract to the presence of pus, can retain it until the involved part of the bronchial tree is full. Then there is apt to be a paroxysm of cough, and if the patient does not drown in his own secretions there is grave danger of a secondary pneumonia. I have narrowly escaped losing patients from each of these complications. In the third place, the nerves remain anesthetized for a period of months, so that the resection of the remaining segments of the corresponding ribs is relatively painless.

We washed out the alcohol we spilt and closed the incision to a Penrose drain, to be left in for twenty-four hours. There was very little damage to the muscles, and the closure in layers was very easy on account of the relaxed condition of the soft parts.

We removed ten centimeter segments of the eighth to the eleventh ribs. At the next sitting, in about a week, we will remove the posterior segments of the fourth to the seventh. At the third sitting, through an incision parallel to and over the ninth rib, we will remove the remaining portions of the four ribs first resected; at the fourth operation, the anterior segments of the upper ribs, and, at the fifth, their lateral segments through a midaxillary incision. The cutting of muscle is reduced to a minimum, and it is remarkable how little deformity and limitation of function result. All our patients have been improved, and there has been no operative mortality in bronchiectasis cases.

CASE OF PULMONARY TUBERCULOSIS

CASE 2.—L. S., a man, aged 33, with a clinical diagnosis of advanced left pulmonary tuberculosis, five years before admission, had developed pulmonary hemorrhage. The sputum was examined and found positive for tuberculosis bacilli. He had been spending the intervening times at various sanatoriums, and had recently been told that his left lung was in collapse. He had had no temperature lately, and his weight was up to normal, but he raised quite a good deal of sputum which occasionally was blood tinged.

Examination: His hemoglobin was 69 per cent. Examination of the sputum showed tuberculosis bacilli present. Roentgen-ray examination of the chest suggested a thickened pleura and possibly a fluid left base displacing the heart to the left, with an immense cavity in the upper left chest. The clinical signs suggested as most likely a thickened pleura at the left base; there were cavity signs in the apex with numerous râles.

Because of the large amount of sputum, which was as high as 7 ounces (207 c.c.) daily, and the fact that the general condition of this patient seemed to be fairly good, we felt that extrapleural thoracoplasty for collapse might be of some benefit to him.

DISCUSSION

DR. CARL A. HEDBLOM: We were dealing here with a case of unilateral pulmonary tuberculosis of several years' standing. There was some question as to the presence of a pleural effusion. In order to rule out that possibility, we aspirated at the point of maximum dulness. As I anticipated, we obtained no fluid. This, together with the roentgenogram, showing marked thickening of the pleura, precluded, I took it, the consideration of pneumothorax collapse. It seemed to me, therefore, a suitable case for extrapleural thoracoplasty.

We had a most stimulating symposium during this meeting on collapse therapy in pulmonary tuberculosis. I believe that, a few years from now, we shall not be so far behind our colleagues in Europe in this form of treatment. At this clinic, we did not attempt a great deal along this line, chiefly because we did not have the facilities for prolonged postoperative treatment which, I believe, these patients should have. It isn't fair to the patient or to the method to turn these patients loose, after operation, without provision for proper supervision and care.

I made the incision and followed the same technic as in the case of bronchiectasis which we just did. In tuberculosis cases, it is especially important, I believe, to avoid penetrating the pleural cavity. I had one patient who developed a miliary tuberculosis a few weeks after operation, and I am not sure that this was not due to operative trauma on the adherent lung. I removed part of the periosteum with the rib, as in the last case.

In pulmonary tuberculosis, it is necessary, I think we all agree, to resect all the upper ribs including the first. I should like to raise the question whether it might not be desirable, in many cases, to remove the whole length of the ribs as we have been doing in the case of patients with bronchiectasis. My impression is that if this can be done safely in bronchiectasis, it should also be possible in the tuberculosis cases. I am just raising the question as to whether some of the patients who are not cured by the ordinary posterior resection might not be cured or at least more markedly improved by a more complete collapse.

Of one thing I am convinced, that is that a two, three, or four stage operation for the resection of the posterior segments is a safer operation than the one stage, and that thereby the indication for operation possibly may be extended to patients too poor risks for a single or a two stage operation.

Dr. Archibald spoke yesterday of the paradoxic retraction of the chest wall following resection. I have noted that the patients who have withstood operation the least have been those who have shown this retraction. A several stage operation will tend to overcome this tendency.

Now we injected the intercostal nerves. There was considerable induration around the nerve and vessels. Here was one enlarged lymph gland which we sent to the pathologic laboratory for examination. It was difficult to isolate the nerve here, imbedded as it was in inflammatory tissue, but here it was. We resected about ten centimeter segments of the eighth to the eleventh ribs and injected each corresponding nerve. We closed the incision to a Penrose drain. The patient was under ethylene anesthesia, and we kept him just as lightly under as possible. He was awake almost as soon as the mask was removed.

The resection of all the ribs posteriorly was completed in three stages. The sputum decreased very considerably in amount, and the patient's general condition improved markedly. A few bacilli were found in some specimens of sputum up to the time of his discharge. Report by letter some weeks after the patient's return home stated that he was gaining but still raising some sputum.

CASE OF TUBERCULOUS EMPYEMA

CASE 3.—T. C. B., a man, aged 36, with a clinical diagnosis of left tuberculous empyema with draining sinus, seven years before admission, developed what was termed "influenza." This lasted three weeks, during which time he was not very sick but had some afternoon temperature and considerable malaise. The temperature persisted, and, five months later, examination of the sputum showed tuberculosis bacilli present. His progress from this time was steadily downward. He developed cough, night sweats and weight loss. He was placed in a sanatorium, and, four years before admission, rather suddenly developed spontaneous pneumothorax. He subsequently developed fluid on this side which became purulent, and a thoracotomy was performed to establish drainage. Since that time, he has had a draining sinus with a small amount of purulent drainage daily.

Examination: His hemoglobin was 74 per cent. with 12,400 leukocytes. Roentgen-ray examination of the chest showed a thickened and retracted pleura on the left, leaving a large pocket. Physical examination of the chest showed a marked deformity of the left chest with a draining sinus over the left anterior chest and practically no expiratory excursion on this side. There were cavity signs over the upper portion of the chest with an occasional fine râle. Examination of the right lung showed a hyperresonance but no signs of activity could be heard. Shortly after admission to the hospital, a tube was put into the empyema pocket by use of a trocar and cannula for the purpose of daily irrigations of the cavity.

DISCUSSION

DR. CARL A. HEDBLOM: This was a case of chronic tuberculous empyema of several years' standing with massive collapse of the lung. The patient, generally speaking, was in poor condition. He belonged to the type in which one might expect a 50 per cent. mortality if one did the Schede operation in one stage. However, we have had a number of patients with tuberculous empyema with large cavities in whom we have succeeded in obliterating the cavity without any mortality by a several stage operation, the number of stages depending on the condition of the patient. We complete the resection of the ribs overlying the cavity first, and then, later, resect the thickened parietal pleura over the residual cavity. Preliminary to operation and between the different operative stages, we irrigate the cavity with surgical solution of chlorinated soda (Dakin's solution) or with saline solution.

In this case, we made a paravertebral incision over the lower ribs. There was overlapping of these ribs. We took the upper one first. There was no objection to leaving the whole of the periosteum behind here. The triangular shape of the cross section of this rib was noted. The outer aspect, normally the wide side, was only half the width of the other two sides. Then we got the lower rib. There was general oozing here which we could perhaps check somewhat with hot gauze. It was important not to lose much blood in these septic and anemic patients.

The tissues were very much indurated, so we did not attempt to isolate the intercostal nerves. That would take a good deal of time, and it was important not to prolong the operation unnecessarily in these cases. The patient lay on his sound side, and with the other lung practically functionless, his respiration was considerably embarrassed. We first injected procain very thoroughly proximally and then a little alcohol distally into the region of the nerve, as indicated by the position of the intercostal vessels. (I have been

impressed with the value of alcohol injection into the intercostal nerves as a means of preventing postoperative pain following plastic operation for empyema. Some of these patients have been out of bed and dressed the day following resection of three or four ribs. One boy recently slipped out and went down town the day after such an operation.) We resected from the seventh to the tenth ribs, and closed the wound to a drain.

It is not possible, in my opinion, to cure all these patients with tuberculous empyema, but I do believe that the majority of them can be cured and many not curable can be greatly benefited. One can collapse the cavity, but in some cases the tissues seem to have lost the power to heal. I doubt the wisdom of attempting surgery in the presence of an active pulmonary lesion. The patient should at least be beyond the acute stage. In most cases, it is possible, by irrigating the cavity, to keep it reasonably clean until the patient's condition improves. Some patients, however, do not seem to tolerate even that. If they continue to lose ground under such treatment, surgery seems out of the question.

CHRONIC LEFT EMPYEMA WITH DRAINING SINUS

Case 4.—L. C., a man, aged 22, who had a clinical diagnosis of chronic left empyema with draining sinus, four years before admission, developed "influenza," which was followed by pneumonia. He subsequently developed a left sided empyema. A thoracotomy was performed, and daily irrigations were instituted. Since that time, he had had a persistent drainage, though irrigations had been continued. His general health had been fair.

Examination: His hemoglobin was 76 per cent. Examination of the drainage from the sinus showed no tuberculosis bacilli or other organisms of consequence. Roentgen-ray examination of the chest showed a retracted pleura on the left, leaving a large pocket.

Dr. Hedblom, at the first and subsequent operations, opened the sinus to allow better drainage, and partially collapsed the cavity by a graded extrapleural thoracoplasty. The patient has gained in weight, and his general condition has improved.

DISCUSSION

Dr. Carl A. Hedblom: The ribs were greatly thickened and deformed, and the surrounding tissue was very much indurated, making the resection difficult. I decided on a several stage extrapleural operation including resection of the first rib because of the very extensive cavity extending to the apex, necessitating a massive collapse, and because the patient's condition did not warrant doing an extensive operation in one sitting. After years of suppuration, these patients are often much poorer surgical risks than their general condition seems to indicate. There may be a myocarditis or nephritis of a much more serious grade than the signs and symptoms reveal. The extrapleural operation enables one to do only as much at each sitting as the patient can safely endure. The fact that the operation is extrapleural also makes it aseptic.

Some perhaps will not agree with me as to the indications for a several stage operation in a case such as this. However, in my opinion, the prolonged convalescence and increased cost incident to a several stage operation are of minor importance in comparison with the greatly increased safety. The first consideration is the life of the patient and the second, conservation of structure

and function. I would not think of doing a big operation and sacrificing a number of ribs for the sake of shortening convalescence, if it could be avoided by other treatment even though time consuming. If I had to choose between six months of treatment and the loss of six ribs, I would take the six months of treatment. I agree with Dr. Yates that we must measure our results in terms of function as well as structure.

The technic here is the same as in extrapleural collapse for pulmonary tuberculosis but more difficult, perhaps owing to the deformity of the ribs and induration present. Resection of the first rib is not as difficult as it is often pictured to be. The important thing is to keep inside the periosteum. If one does that, one can safely resect it from below the intercostal vessels without difficulty or special risk.

I resected the first rib. I handled the resected end with my fingers rather than with lion forceps because it involved less trauma. We inserted a Penrose drain. We tried to inject the intercostal nerves because it is so difficult to isolate them in the presence of so much induration. To attempt to isolate them would consume a good deal of time. The patient was lying on his sound side, and we did not wish to prolong unduly the operation. We inject procain in generous amounts into the region where the nerve lies. We injected alcohol directly into the isolated nerve. Ethylene anesthesia used in combination with local and nerve trunk anesthesia is very satisfactory in these cases. The suturing is naturally very easy, following the relaxation of the tissues caused by the rib resection. We ordinarily get healing by primary intention.

The operative mortality risk in a case such as this is relatively slight. In a recent series of 310 chronic empyema cases including tuberculous ones, those with multiple bronchial fistulas or both conditions combined, those with extensive cavities, such as this one, and many with damaged heart and kidneys, the operative mortality was 1.6 per cent.

CHRONIC RIGHT EMPYEMA

CASE 5.—J. E., a man, aged 29, whose clinical diagnosis was chronic right empyema, had suffered the beginning of his trouble, in 1918, when he developed a bronchopneumonia, which was followed by an empyema on the right side. At that time, three operations were performed for drainage, and the wounds promptly closed. During the period of 1919 and 1920, he was healthy. In 1921, he again developed pneumonia and a recurrence of the empyema on the right side. A tube was inserted for drainage of the empyema, and in three months he was free from drainage and the wound healed. Within ten months, he developed a recurrence of the empyema in the same location, and operative drainage was instituted after which the process apparently healed. Four months later, he again developed an empyema at the same site. This drained spontaneously. In February of this year, he again developed an empyema and a tube was inserted and irrigations performed. Each recurrence of the empyema was preceded by a feeling of malaise, fever and pain at the site of the recurrence, which, in every instance, occurred at the site of the original empyema.

Examination: The patient was fairly well developed. The hemoglobin was 75 per cent., and the urine was normal. Roentgen-ray examination of the chest showed a thickened and retracted pleura on the right, leaving a rather large pocket. Examination showed the entire right chest flat with absent breath sounds and a drainage tube in the sinus situated at about the fifth rib level. Since admission, he had had daily irrigations.

DISCUSSION

DR. CARL A. HEDBLOM: The first step in this operation was to determine the exact size and position of the cavity. One would then be able to extend the incision to the best advantage, and to decide just what type of operation would be necessary. One can determine the size and shape of the cavity fairly accurately by taking a roentgenogram, after filling the cavity with sodium bromid solution, but in case of small cavities with greatly thickened pleura this is not a particularly accurate method. We resected a segment of this rib just above the sinus. The thickening and deformity of this rib was noted. Then we palpated the cavity. It was fairly shallow, but extended beyond reach of my finger. A probe measured its exact extent. We now had our indications. We extended the incision upward between the muscles of the anterior and posterior axillary folds. Then we completely unroofed the cavity, resecting ribs, intercostal tissue and parietal pleura.

In these cases of chronic empyema, we always try irrigation treatment first, using surgical solution of chlorinated soda (Dakin's solution), unless there is a bronchial fistula. In such cases, we begin with saline solution, and it is often possible to use surgical solution of chlorinated soda later. The empyema cavity frequently is reduced in capacity from 50 to 90 per cent. or more by the irrigation treatment. In any case, it greatly improves the patient's general condition and renders the cavity less septic, if it does not actually sterilize it. In this case, the cavity was not much reduced by the irrigation treatment, so a plastic operation was necessary. In many such cases, a rib resection only is indicated. The relaxation of the tissues so produced is sufficient to cause healing very quickly following continuation of the irrigation treatment.

In this case, however, we were dealing with the recurrent type of empyema. Using the word in a loose sense, recurrent empyema is of two types. In most cases, in my experience, there is not a true recurrence. The empyema cavity was present all the time. The sinus healed over, and there was no drainage for periods varying from a few weeks to many years. In many such cases, there is probably a reinfection of the exudate which may have become sterile. In the second type of recurrence, there is probably complete obliteration of the cavity. The patients are perfectly well for periods of years, and then develop an empyema in the same region. Recently, I had the opportunity to observe such a case during the process of recurrence. The patient was a blacksmith who had been perfectly well and working hard for a period of two years following the healing of the empyema. He then developed fever, loss of weight, and had to quit work. He came to the clinic complaining of pain at the right costal margin anteriorly. Roentgen-ray examination of the chest showed only a faint shadow which was thought to be the residue of the old empyema. A tentative diagnosis was made of acute cholecystitis. During two weeks of observation, a second roentgen-ray examination showed a much denser, larger shadow over the old empyema area. At operation, a cavity was drained containing perhaps from 200 to 300 c.c. of pus giving a positive culture. He made a complete recovery. Another patient having the true recurrent type had been entirely well and at work for twenty years before the recurrence developed.

In many of these cases, we find multiple abscesses, sometimes 1 or 2 mm. in diameter, embedded deep in the thickened pleura. I believe these abscesses are the cause of recurrence, at least in some cases. I therefore feel that, in case of a recurrence such as this, it is best to do a complete resection of the parietal pleura.

We resected segments of three ribs well back of the borders of the cavity, taking care to cut the ribs as far back as they had been denuded of pleura. That lessened the chance of osteomyelitis of the rib ends. We cut away the parietal pleura, beveling the edges. There was a little bleeding, which we controlled with the "stick tie." One can tie a vessel embedded in the thickened pleura by this method when it is impossible to tie around the hemostat.

This thickened pleura was examined for tuberculosis. We find that routine examination of excised pleura will show the presence of tuberculous infection in cases in which tuberculosis is not suspected. We also find that assiduous search will reveal tuberculosis in cases in which it is not found at first. Last year, we found that more than 20 per cent. of the cases of chronic empyema were tuberculous. A few years ago, before we became interested in this study, it was under 10 per cent. In some cases, we demonstrate the bacilli in the exudate or by guinea-pig inoculation.

We removed all the sponges. It seems to me almost as easy to leave a sponge in the pleural cavity as in a peritoneal cavity. I have never left a sponge in the peritoneal cavity, but I did leave one once in the pleural cavity. I left a drainage tube in the cavity in one case. The patient at least went home with the tube inside. When he came back with a persistent sinus, roentgen-ray examination showed the tube. I lost no time in getting it out. I wasn't going to let him run off again with that tube. He appreciated very much what we had done for him, but in spite of the fact that he insisted on leaving the first time on such short notice that we did not have an opportunity to make the routine roentgen-ray examination before dismissal, he felt that it was worth something to carry the tube for several months, so we paid him.

Question: Does the clinic carry insurance against such cases?

Dr. Hedblom: Oh, yes. No one can avoid occasional medicolegal complications.

Now, the field was pretty dry. The next question was, Should we leave this wound open or do a muscle plastic and close it tightly? We had the serratus which we had preserved and which we could easily utilize, and, in addition, there was a little of the edge of the latissimus which we tacked down against the pleura. We closed the incision. If the patient developed fever and leukocytosis in a few days, we would have inserted a drainage tube at once.

I like ethylene anesthesia in these cases. I dare say most surgeons use it. There is no stage of excitement during the anesthesia, despite the fact that the patient is very lightly under most of the time. Ethylene seems superior to nitrous oxid in this respect. I like light anesthesia to avoid cyanosis. Cyanosis is bad for these patients who have an impaired circulatory and respiratory function. There is an important investigation being carried on at the University of Wisconsin on the effect of lowered oxygen content which seems of practical importance with respect to cyanosis in anesthesia.

I find I do a somewhat different type of operation in almost every case of chronic empyema. No two of them are quite alike. The operation cannot be standardized because the anatomic and pathologic conditions are so varied.

PULMONARY ABSCESS WITH INFLUENZA

Case 6.—Mrs. A. F. Y., a woman, aged 57, who had a clinical diagnosis of indeterminate pulmonary abscess(?), three years before admission, had developed influenza, after which she had a slight intestinal disturbance consisting of diarrhea with some blood and some pus in the stool. Her recent trouble apparently began in March of this year when she became dull, had

a definite malaise, and some slight fever. A month later, she developed chills followed by a temperature of approximately 101. Ten days before admittance, her fever went as high as 105 with the chills which occurred almost daily. Since she had been under our care, the chills and the high temperature had persisted.

Examination: There was rather high liver dulness extending somewhat above the fourth liver space in the midclavicular line, and the liver edge was definitely palpable. Hemoglobin was 75 per cent., and leukocytes were from 10,200 to 22,000. Blood smears and blood cultures were repeatedly negative, as was examination of the stool for blood and parasites. On chest examination, diffuse râles were heard over the left side. The roentgen-ray examination showed a thickened pleura over the left chest with consolidation of the left lobe, probably an abscess. During her stay here and before, she had never had any sputum, nor had there been other signs of lung abscess. However, because of the roentgen-ray evidence of a consolidation of the left lobe diagnosed possible abscess, and because the patient was definitely going down hill, we felt justified in exploring this area.

DISCUSSION

DR. CARL A. HEDBLOM: I wish to emphasize that here we were confronted by a condition which we had not been able to diagnose. The patient had been having fever of the picket fence type, fluctuating daily from normal to 105, with a leukocytosis up to 40,000. The picture was rather characteristic of liver abscess. The only localizing finding has been a rather indefinite shadow in the roentgenogram of the thorax. On the basis of the symptoms and this finding, one would at once suspect pulmonary abscess, but I have yet to see the first case of pulmonary abscess of any duration without purulent sputum. I was, therefore, very skeptical about finding an abscess, but here was a critically ill patient confronting us, and we were not doing anything for her. Occasionally, we do see a patient who does not present all the findings which we consider essential to the diagnosis of a particular disease condition. I was, therefore, willing to do an exploratory operation for abscess. I was not willing, however, to aspirate through the intact chest wall. We therefore did a first stage operation last week and found the lung not adherent. Had we aspirated and found pus, she would probably by now have an empyema in addition to her other infection. Empyema, in my experience, is the most fatal complication in the drainage of pulmonary abscess. I believe most surgeons feel as I do that aspiration for pulmonary abscess should not be done in the absence of pleural adhesions.

I aspirated, intending if we found pus to open into the abscess with the actual cautery. Before doing the first stage operation, I made a scratch mark at the center of the area, localized by aid of the fluoroscope and physical findings. There also was tenderness to pressure here. Dr. Lemon has emphasized the importance of this tenderness to pressure in the localization of pulmonary abscess, and I have been able to confirm his findings at operation.

I inserted the needle in this area, but obtained no pus or foul air. Dr. Willy Meyer has called attention to the fact that aspirating foul air into the syringe is as significant of having reached the abscess as though one aspirated pus. I have proved that statement to be true in several cases.

In one case which we had a few months ago, I explored for pulmonary abscess in the absence of purulent sputum and found a necrotic area of lung. Examination of a bit of this tissue from the lower lobe showed tuberculosis. In

another case in which there was purulent sputum, microscopic examination of the tissue showed carcinoma. One had to consider these conditions as possibilities, and also interlobar empyema, but the area in question seemed too low for an interlobar process. We broke through the adhesions at the edge of the area exposed. We wished to avoid a pneumothorax and the incident risk of an empyema, particularly in a patient who was already seriously ill. We therefore placed an iodoform gauze pack in the wound and sent the patient back to her room for further observation.

I should like to have suggestions as to the diagnosis in this case.

Dr. Welles: Did you feel the lung with your finger when you opened the chest the first time?

Dr. Hedblom: No, I just exposed the parietal pleura.

Dr. Welles: Did you put a pack against it?

Dr. Hedblom: Yes.

Question: Do you think that an effusion below would cause a temperature?

Dr. Hedblom: Well, I suppose that is possible but I do not believe there is an effusion there.

Question: Could you not get some information from a pneumothorax?

Dr. Hedblom: Suppose we did, and decided it was an abscess. Then what would we do? If there is an abscess at the periphery of the lung without adhesions and one does a pneumothorax collapse, the abscess may break into the pleural cavity producing an empyema. I have seen that happen. If it did not break into the pleural cavity, how would one establish drainage? If the abscess is peripheral, drainage through the chest wall is, to my way of thinking, the most rational treatment. If the abscess is centrally located near the hilum, on the other hand, then the nearest outlet is through the bronchus. Drainage, in such a case, is more difficult and dangerous on account of its proximity to the large vessels.

POSTPNEUMONIC EMPYEMA

Case 7.—Mrs. A. B., a woman, aged 54, with a clinical diagnosis of left postpneumonic empyema, three weeks before admission, felt a pain in the left chest associated with some cough, dyspnea and fairly high fever, presumably pneumonia. She was seen by one of the men from the clinic at her home, some days later. At this time, there was dulness over the entire left chest, with absent breath sounds, and fluid was suspected. An aspiration was done, and a few cubic centimeters of a slightly bloody serous fluid was obtained, which, on culture, showed a hemolytic streptococcus present. There was some edema of both extremities, and the urine showed a considerable amount of albumin present. The patient was too sick to be moved at that time, but, within a few days, she was brought into the hospital. On admission, the pulse was 124, the temperature 104 F., and she was critically ill. Aspiration of the left chest at the base was performed, and 30 c.c. of serosanguineous fluid was obtained. Later aspiration through the posterior axillary line yielded 33 ounces (976 c.c.) of thick pus. Trocar and cannula were then inserted, and drainage and irrigations instituted. The kidney function at this time was only 25 per cent. on a two hour specimen. The blood area was 36 mg. per hundred cubic centimeters, and there was considerable edema of the lower extremities.

She was placed on a restricted protein diet, and fluids were limited. The edema promptly cleared, with improvement of the kidney function. After eleven weeks, the cavity had decreased to practically nothing but a sinus, the size of the drainage tube. In the last week, she had developed slight polyarthritis which had rapidly improved under salicylates and light.

DISCUSSION

DR. CARL A. HEDBLOM: This case was a good illustration of the indication, par excellence, for the closed method of treatment of acute empyema. It was a streptococcus empyema in a patient almost moribund. Had we done an open operation, she would have had added respiratory embarrassment from the penumothorax and that, together with the shock of operation and the sudden evacuation of pus, would probably have carried her off. What we did was to introduce a tube through a stab incision between the ribs under local anesthesia, in the patient's bed. The whole procedure took perhaps two minutes. She didn't realize that we had done anything. Then, as we aspirated off the pus, we replaced it in part by saline solution, which was later withdrawn and surgical solution of chlorinated soda (Dakin's solution) irrigations were started. Altogether, the method represents very slight, if any, additional burden to the patient and is, to my mind, the only justifiable method of draining an empyema in case of a seriously ill patient. I also feel that what is the best treatment for such a patient represents also the least risk to one not so ill.

I probed the sinus and found that it extended forward perhaps 12 cm. I made a long incision parallel to the anterior border of the latissimus. The sinus runs parallel to the direction of the ribs, so by resecting a segment of the ninth rib I was able to unroof the sinus. I was then able to palpate with my finger. There was a very narrow cavity running forward. I had it open in the bed of the rib. How clean the walls of the cavity were was noted; they were covered with healthy looking granulations.

We usually do a minor plastic operation on these acute empyema cases after the cavity has been reduced to from 10 to 30 c.c. capacity. It shortens the time of further convalescence and insures healing of the residual cavity from the bottom. There is always a tendency for the sinus in the soft tissues of the chest wall to heal first, preventing further drainage. This is the most common cause of chronic empyema. There is a minimum of risk in doing a rib resection when the patient is nearly well. We closed this incision, simply placing a drainage tube through the chest wall, leaving an ample opening there.